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DILATATION OF THE AORTA.[1]

By Thomas McCrae, M.D., F.R.C.P.,

ASSOCIATE PROFESSOR OF MEDICINE AND CLINICAL THERAPEUTICS IN THE JOHNS HOPKINS UNIVERSITY, BALTIMORE.

Introduction. As regards the subject of this paper, there is some difficulty in terminology, as the term dilatation of the aorta is variously applied. In this connection it is used to describe the condition in which the aorta is enlarged in its whole circumference, the extent of this varying, sometimes being of the whole arch, sometimes extending to the point where the aorta passes through the diaphragm, and sometimes involving the whole course of the aorta. Is it proper to consider such a condition as an aneurysm? This will depend on the meaning we attach to the term. If we use it in the widest sense as being properly applied to any increase in the size of an artery, this diffuse dilatation would be included. On the contrary, if the term aneurysm be restricted to a tumor containing blood in direct communication with the cavity of the heart or the lumen of an artery, the diffuse dilatation is excluded. The fusiform or globular dilatations of the aorta were designated "true" aneurysms by the older writers. Thoma described them under the designation of aneurysma verum diffusum, and Walshe as peripheric dilating aneurysms. The terms true and false as applied to aneurysms have been used in a confusing way, having been employed in exactly opposite significations. Scarpa used the term "true" to

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designate aneurysms in which all the coats were not present, and "false" to describe those in which all the coats remained and no coagulation had occurred, but the terms were subsequently applied in exactly an opposite sense. It is evident that such distinctions are of no definite value, and the terms false and true are better dropped from the terminology.

In this article the term dilatation is employed to designate a condition which, strictly speaking, probably comes under the term aneurysm as we generally use it, but there is no question that in its clinical aspect it differs considerably both from the saccular and ordinary fusiform aneurysm of the aorta. As Gairdner pointed out, in these cases of diffuse dilatation of the aorta there is nothing that, "clinically speaking, can be definitely called a tumor."

It is surprising how little attention has been given to diffuse dilatation of the aorta, although it has long been recognized. In some text books it is referred to under the headings of chronic aortitis and aneurysm. Morgagni distinguished between the enlargement which involved the whole circumference of the aorta and that which rose from one part, while Scarpa did not regard the enlargement of the whole aorta as aneurysm. Vieussens and Boerhaave also recognized the condition, but it is not always easy to make out whether they are describing a large fusiform aneurysm or the diffuse dilatation.

The first accurate description was given by Joseph Hodgson[2] in 1815, who distinguished the condition from ordinary aneurysm and referred to it as a "preternatural permanent enlargement of the cavity of an artery." By many French writers the condition is termed *maladie de Hodgson*, although some regard an associated aortic insufficiency as an essential part of the picture. Thoma,[3] in 1888, wrote extensively of this form, and gave one of the best descriptions of it. As Osler points out, it is a striking commentary on the difficult yof recognition that, of the cases studied by Thoma,[3] very few had been recognized clinically. It is one of the conditions which has heretofore been more often found in the dead house than recognized in the wards. This is due to various reasons: the condition itself may not give any symptoms, and its signs are overlooked; or signs suggesting some other disease, especially angina pectoris, may cause error; or the signs of associated cardiac disease may dominate the picture.

For this study a series of sixty cases is considered, all of which occurred in the wards of the Johns Hopkins Hospital or were seen in consultation.

ETIOLOGY. There are probably two main groups, one in which the dilatation occurs with an acute infectious disease, notably

[2] Diseases of Arteries and Veins, London, 1815.
[3] Virchow's Archiv, 1888, cxi, 76; 1888, cxii, 259, 383; 1888, cxiii, 244, 505.

rheumatic fever, and the second in which the factors are essentially those which cause arteriosclerosis and aneurysm. In the latter group the essential factor appears to be disease of the vessel wall, with loss of elasticity and subsequent dilatation, probably largely in the nature of a mechanical process. The various factors in this series are as follows:

1. *Age.* Eleven to twenty years, 1; twenty-one to thirty years, 1; thirty-one to forty years, 16; forty-one to fifty years, 16; fifty-one to sixty years, 16; sixty-one to seventy years, 8; seventy-one to eighty years, 2.

Of the 18 patients under the age of forty years, 15 were males (9 white and 6 colored) and 3 females (1 white and 2 colored). The relative preponderance of colored patients among the younger ones is a striking commentary on their tendency to early vascular degeneration. The majority of these 18 patients gave a history of severe muscular exertion for many years; there was a definite history of syphilis in 6 of them, and 7 had aortic insufficiency, while in 5 of the last 10 cases there was a history of heavy drinking. It will be noted that the number is the same in the fourth, fifth, and sixth decades. In regard to the small number in later periods it must be kept in mind that these figures come from a general hospital, and that those with diffuse dilatation in later life rarely have any symptoms from it. It occurs not uncommonly without any symptoms in the aged in almshouses and asylums, and is only recognized at autopsy.

2. *Sex.* The condition occurs most often in males, as might be expected. In this series there were 54 males and 6 females, 4 of the latter being colored.

3. *Color.* The colored race shows a relatively high proportion; of this series, 39 were white and 21 colored (the total medical admissions being about 5 to 1). This is in agreement with the relatively high incidence of aneurysm and sclerotic vascular disease among them, due probably to alcohol, syphilis, and hard work. The total medical patients with aneurysm numbered 399 (white 239, colored 160), so that the relative proportions for aneurysm and dilatation of the aorta are about the same.

4. *Syphilis.* This stands out as an important cause, but probably not so markedly as in aneurysm. There are two principal forms of dilatation influenced by syphilis, one in which the process occurs with an acute syphilitic aortitis, usually seen in young adults, and the other in which the condition results from general chronic changes in the vessel wall. In this series there was a definite history of syphilis in 17 cases and probable in 8 cases. The Wassermann reaction was tried in 8 cases, with a positive result in only 3 cases. This is in such striking contrast to the results in aneurysm that it suggests that syphilis does not play such an important etiological part in dilatation.

5. *Work and Muscular Exertion.* These are factors in dilatation exactly as they are in the production of general arteriosclerosis. Occupations which involve very severe exertion at irregular times, such as that of stevedore, will evidently favor the condition. A sudden increase in the blood pressure must have more influence on the aorta than on any other vessel. In this series 39 of the patients had done hard muscular work, 7 were engaged in business, and 7 did housework. The others had light occupations. Among those who had done hard work, in a large proportion this had involved severe sudden irregular exertion. This is very striking in going over the histories, and an unusual proportion of the men had occupations which involved the lifting of heavy weights.

6. *Alcohol.* How much this is a factor, it is difficult to say. In this series 25 had used alcohol to excess, and 22 were described as moderate users, which is certainly a high proportion.

7. *Acute Specific Infections.* These probably play a more important part than is generally considered, for when we remember how much damage may be done to the aorta in the course of an attack of typhoid fever, it seems probable to suppose that later changes may gradually develop, especially if other factors are added. Whether or not there is a true primary aortitis apart from syphilis is difficult to say, but it seems probable to suppose that it may occur. There is one infection in which dilatation may occur during the acute attack. This is rheumatic fever, in which there may be an acute dilatation of the aorta, usually with aortic endocarditis. There was a definite history of this disease in 13 cases, and in 6 others of an attack of acute arthritis the nature of which was doubtful. A striking feature was the large proportion of the younger patients who gave a history of rheumatic fever, for of the 18 patients under the age of forty years, 9 had suffered from rheumatic fever, several with repeated attacks, which seems a high percentage. As the majority of these had aortic insufficiency, it is a reasonable supposition that acute aortitis occurred, which was followed by chronic changes. Even in young children dilatation of the aorta may occur with aortic endocarditis in rheumatic fever, and it seems reasonable to suppose that this is permanent in some cases. There was a history of typhoid fever in 8 cases, 3 of these being below the age of forty years. In one case, a man aged thirty-three years, the symptoms began during recovery from an attack of typhoid fever, but he had several attacks of rheumatic fever previously. The same condition may occur with acute sepsis. Some writers have suggested that there might be an extension of inflammation from an endocarditis, pericarditis, or pleuritis. It seems reasonable to suppose that if recovery followed an attack of acute aortitis, a chronic condition with tendency to dilatation would be left.

8. *Old Age.* This is only a cause in so far as the passage of years brings sclerotic changes. It is probably then only to be regarded as one of the natural changes.

9. *Malaria.* There are few conditions for which malaria is not given by some one as a causal factor. There does not seem to be any evidence to suggest any association with dilatation of the aorta, but it is constantly repeated by various authors.

10. *Severe Strain or Exposure.* The importance of a factor such as this is difficult to estimate, and any effect is probably only in causing symptoms to appear from a condition already present. There is one striking example in this series:

The patient, a white male, aged thirty-seven years, a ship captain, came complaining of shortness of breath. He had not had typhoid fever, rheumatic fever, or pneumonia. Although he had followed the sea most of his life, there was no history of syphilis, and he had used alcohol very moderately. His illness began one month before, previous to which he had been perfectly strong and well. His ship caught fire, and they had to take to the boats. Naturally he was under a heavy mental strain, and in addition was exposed to bad weather before they were picked up, as a result of which he had a severe attack of bronchitis, with which there was shortness of breath, which continued. He had a well-marked dilatation of the aortic arch, seen with the fluoroscope, with dulness over the manubrium and in both the right and left first and second interspaces, where there was a faint visible pulsation. The heart was not enlarged; the sounds were clear, the second being very ringing. The pulse rate was normal, and there was moderate thickening of the peripheral vessels. His symptoms gradually cleared, and he has been well for some years.

11. *Trauma.* The influence of this is most probably shown in determining the onset of symptoms. In one patient, a miner, aged thirty-nine years, the onset of symptoms followed an injury sus-sustained by being struck on the chest by a dump cart in a mine. He was knocked unconscious and remained so for twenty minutes. On recovering consciousness he had dyspnœa and severe pain in in the chest, which became less after three weeks, but the dyspnœa continued. He was found to have aortic insufficiency, which was thought to have been due to rupture of an aortic cusp. He had been previously a very hard worker in a steel plant, where he carried heavy weights, had drunk heavily, and probably had syphilis, so that there were causal factors other than the trauma.

Morbid Anatomy. The lesions include those of chronic aortitis and secondly the dilatation. This is of varying extent, and may involve only a portion of the thoracic aorta or extend throughout the whole course of the thoracic and abdominal aorta. In the cases of this series coming to autopsy the process did not extend beyond the point where the aorta passes through the diaphragm. The dilatation may end here very abruptly; thus, in one case the aorta measured 8 cm. in circumference at the lower part of the dilatation, and only 4.5 cm. below. The form of the dilatation is

various, and may be spindle, spheroid, or pyriform in shape, or sometimes like a flabby bag. The orifices and part of the vessels given off from the aorta may be involved in the dilatation.

Associated conditions may have a marked effect on the symptoms. Thus, if the chronic sclerotic process has involved the aortic ring, there is likely to be aortic insufficiency in consequence of the valvular changes. If the first part of the aorta is specially involved there may be coronary artery disease. If the orifice of the left subclavian is narrowed the pulse in that arm may be small. One result is of special interest, namely, relative aortic insufficiency due to dilatation of the aortic ring. There were two such cases in this series. In one case a loud aortic diastolic murmur was heard for some time, which disappeared before death, and at autopsy the aortic valves were normal and apparently closed the orifice completely. Probably the decrease in blood pressure, for some days before death, allowed the dilatation of the aorta to diminish and the aortic ring returned to its usual size.

Thrombus formation in the dilated aorta is not common. Osler[1] mentions a specimen in the Museum of McGill University in which the descending thoracic and abdominal aorta was dilated and the abdominal aorta filled by a densely laminated thrombus. Although in the majority of cases the aorta shows marked gross evidences of disease, yet this is not always the case, and the intima may be smooth and apparently normal.

The association of sacculated aneurysm with dilatation of the aorta is mentioned, but no instance was found in the cases of this series which came to autopsy. In one case with general dilatation of the aortic arch there was an aneurysm of the left axillary artery. Thoma figures an instance in which a secondary aneurysm arose through a rupture of the wall of the dilated aorta.

Symptoms. Dilatation of the aorta rarely exists alone, and hence it is difficult to decide which symptoms are due to it and which to associated conditions. When present alone, especially in old people, it may not cause any symptoms, as is proved by the discoveries of the autopsy room. Symptoms due to valvular disease, especially aortic insufficiency, myocarditis, coronary artery disease, and angina pectoris, may be present. The effort to eliminate these as far as possible is made in this study, and it is important to try to decide to what extent we have a definite clinical picture due to dilatation. I believe that in many instances the picture is characteristic, and that the diagnosis can be made with reasonable certainity even before it is confirmed by a fluoroscopic examination. The history of a typical case brings out the general features.

The patient was a male, aged fifty-six years, a merchant by occupation. He came complaining of occasional uneasy feelings

[1] Modern Medicine, iv, 463.

in the thorax, which he described as being rather more than oppression and yet less than severe pain. He was rather apologetic about consulting a physician, as he feared he might be thought to be making too much of a little thing. The family history was negative. In his previous history it was found that the patient had had diphtheria, measles, and typhoid fever. He had not been a specially heavy eater, but had always used alcohol rather freely. For some years he had been working at high pressure, carrying a great deal of responsibility and a heavy load of worry. There was no history of lues.

For about two years he had noted at times, especially with any sudden exertion, such as running for a car, or any unusual excitement, that he had a curious feeling in the chest. This was generally described as being in the sternum or a little to the left of it, although at times it was apparently more marked at the lower end of the sternum, and occasionally it radiated into both shoulders. None of these attacks were at all alarming, and he was always able to go on, although sometimes he had to rest a few minutes before doing so. No one had ever commented on anything strange in his appearance when they occurred. He himself thought it was needless to seek advice, but the continuance of the symptoms rather worried him.

The patient was the picture of robust health; he was well nourished and had a good color. The lungs were clear throughout. The visible cardiac impulse was not marked; the maximum impulse was in the fifth interspace inside the nipple line; the area of dulness was not increased, and except for a soft systolic murmur in the pulmonic area, the sounds were clear. The pulse was 72, of good volume and regular; the blood pressure was 135 mm. On inspection, there was a distinct lift of the manubrium and a visible pulsation in the first and second right and left interspaces. On percussion, there was dulness over the manubrium and to either side. On auscultation, both sounds were heard at the base, the second with a marked ringing, amphoric quality, which could be made out over a rather wide area. The fluoroscopic examination showed a diffuse enlargement of the whole aortic arch, and a shadow could be seen both to the right and left of the sternum, continuous with that of the heart. This patient has been under observation for nearly four years and no material change has occurred, except that the "attacks" have decreased and are now infrequent.

Clinically there are several groups of cases, which usually can be separated without much difficulty:

1. *Latent Cases.* Absence of all symptoms is especially likely to occur in old people, and the condition is often found unexpectedly post-mortem. In this series there were nine patients in whom the condition was recognized only by the routine examination, as they made no complaint of any symptoms which suggested disease of the cardiovascular system. One was admitted for diabetes, one for chronic alcoholism, one for organic disease of the spinal cord, one

for lipoma, one for diarrhœa, and the others for indefinite complaints. In all the diagnosis was confirmed by the fluoroscopic examination.

2. A group in which *cardiac symptoms*, suggesting failure of the heart muscle, predominate. In the cases with marked signs of valvular lesions, aortic insufficiency predominates. The picture is often that of cardiac failure, which is the final stage in the majority of cases, not because of the dilated aorta, but on account of the associated condtions.

3. A group with features much like those of *angina pectoris;* in fact, sometimes it may be difficult to say that angina is not present. There were fifteen patients in this series in whom the pain might be described as anginoid, and others gave a history of it. In all of those who had the attacks of thoracic pain while under observation it was noted that the severity was less than that usually seen in angina pectoris.

4. A group in which the symptoms are those of the condition in the aorta itself, the heart being practically normal. These patients often give a history of symptoms which suggests cardiac disease.

Complaint. This usually suggests some circulatory disturbance. The most frequent is shortness of breath and dyspnœa, usually not constant unless there is a marked cardiac condition present. Next in frequency is the complaint of thoracic pain of varying degree. Œdema of the feet and legs, which in several occurred without the heart showing any signs of disease, is fairly common. Weakness is rather a frequent complaint, and loss of weight, cough, palpitation of the heart, syncopal attacks, giddy feelings, hoarseness, thumping in the chest and neck, change in the voice, and indefinite pains were also mentioned by some patients. Only one patient complained of difficulty in swallowing; this was verified by observation and occurred quite frequently. That certain patients had no symptoms referred to the vascular system has to be remembered in regard to the figures.

Onset. This was gradual in 41 and sudden in 10 (9 had no symptoms); in 3 of the latter it followed unusual exertion, in 1 injury, in 1 severe mental strain and exposure, in 1 the change to a high altitude, and in the others there was no definite cause.

Dyspnœa was the most common symptom, and occurred in 42 cases. Its severity depended largely on the state of the heart, and when this was normal it was usually brought on by special exertion or excitement. Cough accompanied it in 16 cases, in 3 with spitting of blood. A striking point in several cases was that the dyspnœa was quite out of proportion to anything that could be found to account for it.

Œdema of the feet and legs occurred in 16 cases. In several of these the heart seemed normal in every way, and, as a rule, the blood pressure was low.

Pain was a prominent feature in 20 cases. In 6 of these it was referred to the chest generally without any special localization; in 4

it was referred to the left chest; in 9 it was referred to the heart, and in 5 of these it radiated to the shoulder and arm (3 to the right, 1 to the left, and 1 to both arms); and in 1 it was on the right side of the sternum. The attacks of pain were usually not very severe, but sometimes continued off and on for some hours, and generally were not accompanied by any change in the patient's appearance. In only one case was there any note of sweating with the attack of pain. Walking against a strong wind, sudden exertion, excitement, and overloading of the stomach were all given as causal factors, by which angina pectoris is suggested.

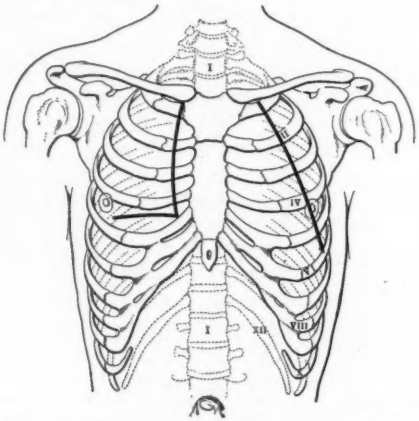
PHYSICAL SIGNS. With the frequent association of various cardiac conditions it is natural to have signs due to them, but these require no discussion and only features of dilatation are considered. A striking condition is marked fulness of the veins, especially those of the neck, upper thorax, and arms, the first named usually being the most dilated. The visible arterial pulsation is sometimes very diffuse, and often very marked over the carotids and subclavians. In several cases it was much more marked over the right than over the left carotid. The subclavians seem to be lifted higher than normal by the dilatation of the aorta and may be seen above the clavicles. There may be a visible swelling, as in two patients in whom a pulsating mass the size of a hen's egg was visible at the inner end of the right clavicle and in the episternal notch. With the enlargement of the aorta it would be expected that pulsation in the episternal notch would be frequently seen, and such is the case, as in 22 cases it was well marked.

A most important point on inspection is pulsation in the first and second interspaces. This is usually best seen with the patient lying down and the examiner with his eyes about on a level with the thorax; good light is essential, and the inspection should be from various positions in regard to the patient. An impulse may be seen in one interspace only or in any combination of the first and second right and left interspaces. The second right is the most frequent situation, then the second left, first right, and first left interspace. This visible pulsation, as a rule, is more diffuse and not so localized as in saccular aneurysm. In some cases it is more evident when the patient is sitting than when lying. It can rarely be felt distinctly, and does not give the feeling of being more forcible than the cardiac impulse; in fact, the pulsation in dilatation is usually more evident on inspection than on palpation. It does not lift the palpating finger, as may occur in aneurysm. There was a definite lift to the manubrium in 26 cases; this was usually better seen than felt. In 5 the absence of any visible heaving was noted, and in the remainder the point was not noted. In two cases the manubrium was prominent and there was a distinct lift of it and the inner end of both clavicles; in one, of the manubrium and right clavicle. In one the inner end of

the right clavicle was lifted. In some cases it was noted that there was a widespread visible heaving, but no well-marked local impulse.

Pulsation in the back in the left interscapular space was observed in one case only. In this case it disappeared after a period of rest, which usually results in a diminution in the extent of the visible pulsation in front.

Palpation. In a number of cases the aorta can be palpated in the episternal notch and the subclavian arteries above the clavicles. In one case there was a distinct pulsating tumor projecting above the inner half of the right clavicle and in the episternal notch; the finger could be passed between it and the clavicle. The heaving of the manubrium could be felt in 6 cases. The contrast between the marked *visible* lift of the manubrium and the slight *palpable* lift is often very marked. The shock of the second sound was felt at the base in 9 cases. A diastolic thrill was felt over the manubrium in 4 cases, and only in the aortic area in 4. In 1 case there was a marked diastolic thrill over the right sternoclavicular joint, the manubrium, and the upper half of the sternum.



The area of dulness in dilatation of the arch of the aorta.

Percussion. The results of percussion are perhaps the most important in the recognition of dilatation, and the finding of dulness over the manubrium in a routine examination may suggest the diagnosis. In 55 cases of the 60 there was distinct dulness over the manubrium. In 2 cases its absence was noted, 1 being with marked emphysema, and in 3 cases there is no note. The area of dulness was usually continuous with the cardiac dulness, and generally extended some distance outside the edge of the sternum, as a rule, both in the first and second interspaces. In 28 cases it was marked in the first and second right and left, in 7 in the first right and left, in 2 in the

first left, and in 2 each in the first and second right and first and second left interspaces. The width of the area of dulness varies somewhat, the usual transverse measurement being about 7 or 8 cm. The widest was 14 cm. in the first interspace, and there were several with an area of dulness measuring 10 cm. across. As a rule, the dulness is more marked to the left of the sternum than to the right. The usual area of dulness is shown in the accompanying diagram. Changes in some extent of the area of dulness occurred in several cases, some increasing and some decreasing. In some patients who have been followed for years a definite increase in the extent of dulness has been noted. In one patient, who was bled 500 c.c. with marked benefit to symptoms due to cardiac failure, no decrease in the area of aortic dulness was made out. The occurrence of an area of dulness, as described by Potain, extending from the manubrium in a segment of a circle to the right, with a shape like the "crest of a fireman's helmet," has not been made out.

Auscultation. When the second sound is present it often has a distinct quality at the base which is the most characteristic sign on auscultation, and in several instances first suggested the diagnosis. This is a bell-like, ringing, amphoric quality, very distinctive when once heard, and quite different from the accentuation found with high blood pressure. It has been given various descriptions, "clangoreux" by de Mussy, "bruit de tabourka" by Potain, "timbre métallique," etc. This sound may be heard over a wide area and with especial distinctness over the manubrium and in the neck over the carotids. It was well-marked in 12 of 22 cases in which the second sound was clear. In 5 cases with aortic insufficiency the second sound had this quality, and the diastolic murmur had somewhat the same. In 2 cases of aortic insufficiency in which the second sound had entirely disappeared, the diastolic murmur had this amphoric, musical quality. When aortic insufficiency is present the diastolic murmur may be heard with great distinctness over the manubrium and at the episternal notch.

Pulse. In the cases with no decided cardiac change there was no striking peculiarity. The rate was not particularly rapid; in about one-third of the cases it did not rise above 80, and in the same number was not above 100 as a rule. In 4 cases the rate was persistently below 70, in 1 being usually about 50 per minute.

Blood Pressure. It might be expected that this would be high and such a statement is made in the literature, but the results of this series show the contrary. Of course, in a condition which is so likely to be associated with other vascular lesions, there are many factors which influence the blood pressure. However, in the cases in which the aortic dilatation was the prominent factor the blood pressure was low. This may seem surprising when we consider the common sclerotic changes, but the alteration in the aorta must have considerable effect. The large vessels by their elasticity tend to convert

the intermittent force of the heart into a continuous pressure. But when the aorta is dilated and has lost its elasticity, the blood is forced into a more or less rigid reservoir and a low pressure is the result. Among 48 cases with records, in 35 the average systolic pressure was below 140 mm. (Riva-Rocci instrument, broad arm-piece). In 3 of these it rose temporarily to higher figures. In 3 patients it varied from 70 to 95, never being above the latter figure, and with an average of 80. One of these had acute endocarditis on an old chronic process, with which he died. The other had quite marked arteriosclerosis with aortic insufficiency, and was discharged from the hospital in fairly good condition. Of the remaining cases, 9 varied from 125 to 180, and the others from 150 to 260. In the majority of these the higher figures were only for short periods, the pressure falling rapidly, in one case from 200 to 130, where it remained. Practically all the patients with the higher figures had cardiac hypertrophy; in the patients without marked changes in the heart the pressure was usually between 120 and 135. Crises of hypertension described by some writers were not observed in this series. Several patients with chronic nephritis (urine of low specific gravity, containing a very slight amount of albumin and a few casts), had a low blood pressure.

In 9 cases in which there were records of both the systolic and diastolic pressure, these showed a fairly steady difference of about 30 mm. Hg. This was practically the same whether the pressure was high or low.

Fluoroscopic Examination. This is a most important part of the examination, and in doubtful cases is usually the deciding point. Dr. Baetjer, who has had a large experience in thoracic fluoroscopic examinations, gives the following description: "Dilatation of the aorta is marked by an increased width of the aorta. In normal conditions fluoroscopic examination reveals only the bend of the aorta projecting to the left of the sternum and just beneath the clavicle, the ascending and descending portions being invisible behind the sternum. In dilatation there is a shadow projecting to the left of the sternum and extending downward to be lost in the shadow of the heart. This shadow may vary in size and in extreme dilatation may project to the right of the sternum. This shadow persists between pulsations, showing that there is a true dilatation of the aorta. In neurotic individuals and when the blood pressure is low we frequently find the aorta, in pulsation, extending beyond the sternum, but between pulsations it invariably recedes behind the sternum and is lost to view." The degree of visible pulsation varies, but in the majority there is no great difference in the extent of the shadow during systole and diastole. In one patient with quite marked dilatation it was noted by Dr. Baetjer that the shadow to the left of the sternum did not vary, but that to the right did. This may have been due to the greater direct force of the flow

from the ventricle on the first portion of the arch. In several instances it was noted that the shadow of the aorta extended unusually high in the thorax. Dr. Baetjer tells me that in several patients the position of the heart in the thorax seeemd more horizontal than normal.

Pressure Effects. These are interesting in view of their frequency and importance in the symptomatology of saccular aneurysm of the aorta. In diffuse dilatation they are less frequent and less serious.

(*a*) *Vascular System.* Fulness of the veins of the neck, upper thorax, or arms occurred in about one-third of the cases. In 6 cases the radial pulses were unequal, in 4 the left being the larger and in 2 the right. The blood pressure was unequal in the brachials in 3 cases, in 2 that in the left brachial being the higher. The difference varied from 13 to 18 mm. Hg. In one case the vessel with the larger volume had the lower blood pressure. The difference in the radial pulses may be due to some obstruction at the beginning of the innominate or subclavian artery, or the innominate or subclavian artery may be involved in the dilatation.

(*b*) *Respiratory Tract.* A tracheal tug was noted in 12 instances, in 4 of which it was quite marked for some time after admission, but with improvement in the general condition it disappeared. In one patient the tracheal tug was present on several occasions on admission, but disappeared under rest, and coincident with this it was noted that the area of aortic dulness had decreased. In some patients there was dulness at one or both bases, but this may have been due to the state of the heart. In 3 patients there was recurring hydrothorax on the right side, for which repeated tapping had to be done.

(*c*) *Nervous System.* There was inequality of the pupils in 9 patients. In 3 the left was the smaller, in 5 the right, and in 1 there was no note. The usual opinion has been that this was due to pressure on the sympathetic, but alterations in the circulation may be more important. There was pressure paralysis of the left vocal cord in 3 patients, and in 5 others the voice was hoarse and the cough had the "goose" quality. One patient has been under observation since 1905, at which time he had complete recurrent laryngeal paralysis of the left vocal cord, which has continued to the present. The first symptoms were two years before his first admission, when he had pain in the upper thorax, with suffocating feelings and a marked change in the voice.

In some cases the pain or numbness felt in the shoulders and arms was persistent. One patient had constant pain in the left shoulder, which occasionally radiated down the arm and to the scapula. In one patient pressure over the fifth, sixth, and seventh dorsal spines caused severe pain. The same patient had pain in the heart and left shoulder on firm pressure over the precordium.

(*d*) *Bones and Muscles.* There was no instance of perforation of the thoracic wall or erosion of the vertebræ.

(*e*) *Alimentary Tract.* Dysphagia was present in one patient. No attempt was made to prove the presence of obstruction.

It is rather surprising, perhaps, to find the pressure signs occurring as often as they do, although, of course, with less frequency than in aneurysm. They that should occur is not surprising in view of the anatomical conditions. In a previous paper on this subject[5] the view was taken that the pressure effects were comparatively few, but the study of subsequent cases has rather modified this view. Still the pressure effects are rarely serious.

Associated Conditions. Thickening of the palpable arteries was found in the great majority of the patients, and in 39 cases it was noted as being marked. In 5 there was calcification of the vessels. Aortic insufficiency was present in 27 cases, in 18 with mitral insufficiency, in 3 with aortic stenosis and mitral insufficiency. Mitral insufficiency without an aortic lesion was found in 4 cases, all having marked arteriosclerosis. The heart was practically normal, so far as could be made out, in 18 cases; in 5 others there was marked hypertrophy, and in 4 others slight hypertoropy without any valvular lesion.

Relative Aortic Insufficiency. This is rare under any circumstances, but dilatation of the arch of the aorta should favor its production. There were 2 cases in this series in which the signs suggested this condition.

The first patient was a colored female, aged thirty-two years, admitted March 25, 1898, with dyspnœa and œdema. She had marked fulness of the veins of the neck and upper thorax. The heart was enlarged to the left and there was a heaving impulse over the manubrium, with dulness, which extended into the first interspace on both sides. There was a well-marked systolic murmur at the apex and a definite diastolic murmur in the aortic area. The second sound could be well heard over the manubrium and in the neck. Later there was a diastolic shock at the base. The pulse varied from 70 to 80; it was not specially collapsing, and the pulse tracing did not suggest aortic insufficiency. The presence of the aortic diastolic murmur was noted by Dr. Osler. The fluoroscope showed dilatation of the arch. She was admitted six months later with marked dyspnœa and œdema. There was marked pulsation in the episternal notch, but the heaving of the manubrium was not as marked as on the previous admission. Over the body of the heart the second sound was much accentuated, but no diastolic murmur could be heard. She died the day after admission. The autopsy showed arteriosclerosis, hypertrophy of the heart, pulmonary thrombosis, and dilatation of the arch of the aorta, with uncom-

[5] Canada Lancet, 1907–08, xli, 821.

pensated degeneration in the intima and media. The aortic and mitral valves were normal. The aorta was much dilated and thin, measuring 9 cm. in diameter above the valves. It showed many areas of nodular thickening, and three areas about 1.5 cm. in diameter, which were very thin.

The second case was a man, aged sixty-four, who was admitted with dyspnœa and swelling of the feet. The heart was much enlarged, with a soft systolic murmur at the apex and a definite diastolic murmur in the aortic area. The second aortic sound had a ringing amphoric quality. Within a week the diastolic murmur had disappeared, to reappear later. Four months later he was admitted again with a curious continuous murmur best heard at the aortic area. At times it had a systolic intensification, but was very variable and markedly altered by changes in the patient's position. The diastolic murmur was not heard on later examinations.

The infrequency of relative aortic insufficiency is well recognized, and the experience of this series does not support the view expressed by Balfour,[6] that "when once dilatation of the ascending aorta is produced, the secondary development of incompetence of the aortic valves is only a question of time." Some of these patients have been under observation for years and an aortic murmur has never been heard.

Renal System. This showed changes in the majority of cases. A number had chronic nephritis; others had albumin and casts or albumin alone.

Blood. This showed no changes of any moment. The count was about normal in the majority, a few showing secondary anemia.

Intestinal Hemorrhage. One patient, a colored man, aged thirty-three years, had several large hemorrhages from the bowel, for which no cause was found. This is interesting in connection with the observation made by Albers, of Bremen, and quoted by Dr. Osler,[7] in which hemorrhage of the bowels occurred with dynamic dilatation of the aorta.

Course. In so far as dilatation of the aorta is concerned, apart from other cardiovascular lesions, there seems no reason why it should not continue indefinitely. It is the associated conditions which are of moment in the prognosis. One patient in whom the condition was recognized in a routine examination has gone for five years without any evident change, and without any symptoms which might be referred to the condition of the aorta. Serious accidents, such as occur in saccular aneurysm, were not noted in this series. There was no instance of rupture of the aorta, although it seems possible that this might occur, or of dangerous pressure

[6] Clinical Lectures on the Diseases of the Heart and Aorta, London, 1882.
[7] A System of Medicine, Allbutt and Rolleston 1909, vi, 669.

signs. The mode of life must have considerable effect on the outlook, and patients who can live a quiet regular life may go on indefinitely. Unfortunately in many of the patients the occupation is an important factor in the etiology, and they are often unable to obtain easier work. Occupations involving sudden severe exertion have a very detrimental effect. In the majority of cases it is probable that the condition of the first inch of the aorta will largely determine the outlook. With serious disease of the aortic orifice or interference with the coronary circulation, the prognosis is grave.

DIAGNOSIS. It is important to recognize the frequency of the condition and to be on the watch for it. The complaints usually suggest disease of some other organ, and the physical signs may not attract attention—they have to be looked for. There are two groups of cases in which the diagnosis may be suggested. In one the history and symptoms suggest cardiac disease, but on examination no disease of the heart is found. In the other group the history of thoracic pain may suggest angina pectoris, but careful scrutiny does not bear out this diagnosis. In many cases the dilatation of the aorta is accompanied by cardiac disease, and it is easy to rest satisfied with the recognition of the most obvious process. Of the physical signs, the visible impulse seen over the manubrium and in the adjoining first and second right interspaces, the area of dulness, the curious amphoric quality of the second sound, if this be present, and the fluoroscopic examination give the greatest aid.

Various other intrathoracic conditions have to be considered, the tendency being rather to regard dilatation as being one of these than the contrary.

1. *Saccular Aneurysm of the Aorta.* The view may be taken that there is no need to attempt to distinguish between saccular aneurysm and diffuse dilatation of the aorta. This, however, is not correct, because the treatment is entirely different. To attempt to wire a diffuse dilatation of the aorta could not possibly be of any advantage and would do harm; while as regards a prolonged period of rest, it is doubtful if this is indicated in diffuse dilatation. The outlook is also quite different, being much less favorable in saccular aneurysm. The points in the differential diagnosis are as follows:

(*a*) *Pulsation.* As a rule, the visible pulsation is more diffuse and widespread in dilatation, and an important point is the difference on palpation. The impulse in the interspaces in dilatation may be readily visible, but cannot be felt, while in the sacculated aneurysm it is more punctate and readily palpable. Very often the pulsation of a saccular aneurysm is more forcible than that of the heart. The heaving impulse of the manubrium is not readily felt in diffuse dilatation and the impulse in the interspaces may not be palpable.

(*b*) *Percussion.* The area of dulness in dilatation is more or less regular. It is found over the manubrium and perhaps on both sides of it, usually rather more to the left. As a rule, it is continuous with

the cardiac dulness, whereas in saccular aneurysm very often an area of resonance is found between the cardiac dulness and that of the aneurysm. In only one instance of this series was dulness made out in the interscapular region and this was only temporary, disappearing under rest.

(*c*) *Auscultation.* The curious quality of the second aortic sound, already referred to, is very significant when present. The wide area over which the second sound and the murmur of aortic insufficiency, if present, is heard, may be an important point.

(*d*) *X-ray Examination.* This gives information of the greatest value, and in a doubtful case must be the deciding test. Naturally its value depends on the experience of the observer. The possibility of atypical pictures due to some unusual position of the aorta must be kept in mind.

(*e*) *Pressure Effects.* These are decidedly less in dilatation than in aneurysm, but, as noted in the discussion of the physical signs, they may be marked. Severe pressure signs, especially thoracic pain, speak against dilatation and for aneurysm. Certainly, continuous pain is rare in dilatation and the evidences of severe pressure, dyspnœa, stridor, etc., are apparently unknown. The variation in the pressure signs in dilatation has been noted.

2. *Intrathoracic Tumor.* This may give difficulty, but the pressure signs of tumor are likely to be more marked. The visible pulsation, usually so characteristic of diffuse dilatation, is not likely to be present. In a case in which there is doubt from the physical signs the examination with the fluoroscope will usually settle the question at once.

3. *Malposition of the Aorta.* If the aorta is unusually twisted, and lies further to the right than usual, there may be visible pulsation to the right of the sternum and an increase of dulness, or in patients with scoliosis, the vessel may be out of the normal position. The same may be seen in some of the thoracic deformities due to rickets with displacement of the heart and aorta, or in instances of marked changes with shrinking of one side of the thorax. The general features should suggest the diagnosis, and if there is doubt the *x*-ray examination would decide without difficulty.

In the distinction between saccular aneurysm and dilatation or malposition of the aorta, the continuance of the signs and symptoms without any great change has to be kept in mind. While a saccular aneurysm may show but little change during a considerable period, as a rule it tends to be progressive. In the absence of cardiac features, diffuse dilatation may apparently go on indefinitely without any marked change.

4. *Angina Pectoris.* The complaint of thoracic pain may suggest this diagnosis, and until the patient can be observed for some time a decision may be difficult. There is no reason why pain similar to that of angina should not occur in dilatation, especially in view

of the explanation which ascribes the pain of angina to disease of the first part of the aorta. There are certain features which are generally distinctive. The pain in dilatation is rarely so severe as in angina, is often described as more a feeling of oppression than of pain, may persist for longer periods, occurs not infrequently on the right side, and is sometimes felt in both arms. This last should always suggest dilatation. The pain in dilatation does not often seem to be caused by exertion, in fact several patients found that moderate physical exertion relieved the pain. In only one case did sweating occur with the pain. In no case, so far as known, has death occurred during an attack of pain.

5. *Cardiac Disease.* As Hodgson emphasized, the symptoms of dilatation of the aorta are often regarded as due to cardiac disease. When this does not co-exist a careful examination should prevent any mistake.

TREATMENT. Any associated vascular condition, valvular or muscular insufficiency, or myocarditis, demands its usual treatment. When present, so far as treatment is concerned, they give the usual indications. As regards dilatation itself, the main points are as follows:

Rest. There is no universal rule, and the question must be decided for each patient. If there is pain, dyspnœa, distress, or œdema of the feet, a period of absolute rest is advisable until relief is obtained. But rest should not be too prolonged, and in an ordinary case there does not seem any advantage in more than two or three weeks. Prolonged periods of physical rest are not advisable, and rest from worry and the strain of daily troubles seems more important. A long vacation is advisable if it be possible. In the laboring classes, to which so many of these patients belong, the problem is more difficult. Occupations which involve severe strain should be given up and every effort made to have the patient employed at some lighter task. When the patients are free of symptoms a certain amount of exercise is an advantage, if the associated conditions do not forbid. Several of the patients in this series find golf an excellent form of exercise.

Diet. This should conform to the general rules for arteriosclerosis. Reduction in the amount is most important, and meat especially should be limited in amount. Alcohol is best omitted, and fluids should not be taken in large amounts.

Bowels. It is most important to keep these freely open, for constipation seems to be especially harmful. Such purgatives as senna, aloes, cascara, and phenolphthalein may be given, and an occasional mercurial (nothing is better than 5 grains of gray powder) may be given at night followed by a saline in the morning.

Medicinal. There is no drug which can have any marked effect on the aorta, for the damage is done. Potassium iodide should always be given and small doses (3 to 10 grains) are probably best, over considerable periods of time. The vasodilators are usually

not indicated; if high pressure is present they may be tried cautiously. Digitalis is not indicated for the aortic condition alone and may do harm. For the attacks of pain—if severe enough to demand treatment—codeine may be given by the mouth; morphine is rarely required, but should be given if the pain is very severe. Some patients, even with a low blood pressure, find that nitroglycerin ($\frac{1}{100}$ to $\frac{1}{50}$ grain) gives almost immediate relief, and there can be no objection to its use. Others find relief from a dose of aromatic spirit of ammonia (℥i) with spirit of chloroform (♏xv).

In general, it may be said that a quiet even life, with avoidance of physical and mental strain, a diet limited especially in amount, open bowels, and meeting special indications when necessary, are the main points. With these we may hope to give the patients in whom the heart is not markedly affected many years of comfortable life. When cardiac breakdown appears, it must receive proper treatment.

CONCLUSIONS. 1. Dilatation of the aorta, considered apart from aneurysm, is relatively common and probably occurs more frequently than aneurysm.

2. Two main etiological factors stand out—(*a*) acute infections, notably rheumatic fever, and (*b*) those which cause general sclerotic changes.

3. The symptoms often suggest disease of the heart itself.

4. The physical signs are often very definite, of which visible pulsation in the upper interspaces, dulness over the upper sternum and adjoining interspaces, and the fluoroscopic examination are the most important.

5. Pressure signs are comparatively common, but are rarely severe.

6. The diagnosis can usually be readily made from aneurysm.

7. The outlook as regards dilatation is good.

HEART BLOCK WITH AN INDICATION OF GENUINE HEMISYSTOLE.[1]

BY ALFRED STENGEL, M.D.,
PROFESSOR OF CLINICAL MEDICINE IN THE UNIVERSITY OF PENNSYLVANIA,
AND
WILLIAM PEPPER, M.D.,
ASSISTANT PROFESSOR OF CLINICAL PATHOLOGY IN THE UNIVERSITY OF PENNSYLVANIA,
PHILADELPHIA.
(From the William Pepper Laboratory of Clinical Medicine.)

THE case of heart block which furnished the basis for the observations which we wish to report was not of such exceptional character that a separate publication of the clinical features would be

[1] Read at a meeting of the Association of American Physicians, Washington, D. C., May 3 and 4, 1910.

of value; but the numerous tracings which were obtained furnished some interesting information and by lucky chance one of these proved of unusual importance in throwing some light on the possibility of true hemisystole. We shall give only the outlines of the history and clinical aspects of the case and then proceed to the discussion of the more interesting features.

The patient was an Italian, aged thirty-nine years, a cabinet maker, and married. Three years ago pains in the epigastrium occurred. One year ago he had a severe attack of the same pain with an attack of syncope, but noticed nothing wrong with his heart. He had a fainting attack in August, 1908. Ten days before admission to the University Hospital he had epigastric pain, and was in bed for a few days. On returning to work the pain recurred at once, and he then noticed that it came with each beat of the heart and that the heart was beating very slowly. He went home, where he had several attacks of faintness, vertigo, and vomiting.

When twenty-three years old he had a suppurating bubo and a sore on the penis. No secondaries followed, but after leaving the hospital where he went for treatment several of his teeth fell out. He has four living healthy children; his wife had one miscarriage before the four children were born. He has always been a healthy man, although he has not felt quite so well during the last three years. He has some pain along the edge of the tibiæ, especially in winter. The Wassermann reaction was positive.

The heart was enlarged one and one-half inches to the right of the sternum, but not beyond the left midclavicular line. A long systolic murmur was heard, and a dull second sound. When admitted, on February 5, 1910, at 6.30 P.M., the pulse rate was 62. The next morning it was 76, but it rapidly fell to 22 by noon, and did not rise above 34 until 6 A.M. on February 9.

The features that we wish to discuss are the following:

1. That although for a time complete block existed and later partial heart block, during the patient's stay in the hospital no Stokes-Adams syndrome was present.

2. That the complete block, after the administration of atropine, promptly changed to an incomplete block with a 3 to 1 rhythm, then to a 2 to 1 rhythm, and later to a normal rhythm.

3. That the 2 to 1 rhythm and the normal rhythm for a time alternated one with the other, and a number of tracings were obtained showing this change while the tracings were actually being made.

4. That it furnished indications of genuine hemisystole during the transition of the normal rhythm to a 2 to 1 rhythm.

5. That from this same tracing it could be demonstrated that the origin of the *c* wave is the right ventricular contraction, rather than the carotid pulsation.

6. That the *a-c* interval is longer in such cases as this during normal rhythm, while it may be normal in length during partial block.

7. That the auricular rate becomes faster when the normal rhythm changes to a 2 to 1 rhythm, and slower when the opposite change occurs.

8. That explanations were suggested for the increased or decreased auricular rate during block and normal rhythm and for the irregular ventricular rhythm during complete block.

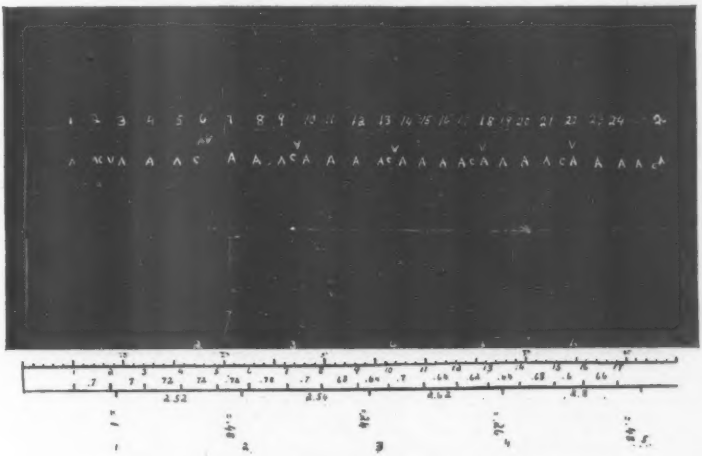


FIG. 1.—Tracing made February 6, 1910. Complete block.

1. A study of tracings 1, 2, and 3, made on February 6 and 8, shows that the block was complete, and that during this time the radial pulse fell as low as 17 to the minute, and pauses were noted between ventricular contractions of as much as 5.4 seconds. Several times 7 auricular contractions were observed between 2 ventricular ones, and yet not the slightest evidence of any Stokes-Adams syndrome was noted during the patient's stay in the hospital. During the phase of complete block the average ventricular rate from 35 measurements taken from 8 different tracings was 19.2 per minute, while the average auricular rate from 174 measurements was 80.7 per minute, a little more than four times as many to the minute.

2. A study of tracings 2 and 3, made at 1.30 P.M. on February 8, shows a complete block at that hour, and tracing 4, made 4 hours later after two hypodermic injections of atropine had been given —one at 1.30, of $\frac{1}{100}$ grain, and another at 4.30 of $\frac{1}{75}$ grain— shows that the complete block has changed to an incomplete one with a 3 to 1 rhythm. In tracing 4 the auricle was beating about

81 times per minute and the ventricle 27. Atropine was then given by the mouth every third hour, $\frac{1}{200}$ grain for two days, then withheld for four days, and then given again for a week. Tracings on the ninth day after the change from complete block to incomplete block, with a 3 to 1 ryhthm, showed a 2 to 1 rhythm, and thereafter up to the present date the tracings have shown either a 2 to 1 rhythm or no block at all. He has apparently had no return of the complete block since the two hypodermic injections of atropine on February 8.

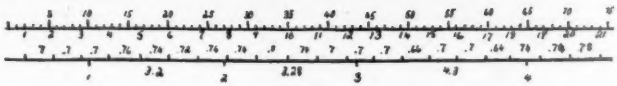


FIG. 2.—February 8, 1910. Before atropine was given. Complete block. Irregular ventricular action.

Figs. 3, 4 and 5 can be well considered together. On examining tracing 5 it will be observed that the normal rhythm changes suddenly to a 2 to 1 rhythm, brought on possibly by a deep breath that the patient had taken about seven seconds before, though more probably coming on spontaneously, as at this time the 2 to 1 rhythm and the normal rhythms frequently alternated. It will be seen that the first six *a* waves are followed, as they should be, by a *c* wave and accompanied by a wave in the radial tracing, but that the seventh *a* wave is not followed by a *c* wave, although accompanied in the radial by a wave exactly like the preceding one. The eighth *a* wave is followed by a *c* wave and accompanied by a wave in the radial, the ninth *a* wave is not followed by a *c* wave and is not accompanied by a wave in the radial, and from here on the 2 to 1 rhythm which has been started persists. Now, how are we to explain why the seventh *a* wave is not followed by a *c* wave, although accompanied by a wave in the radial tracing? There being an *a* wave shows that the right auricle con-

tracted, and there being a wave in the radial shows that the left ventricle contracted following the auricular contraction, but why is there no *c* wave? If the *c* wave is caused largely by the carotid

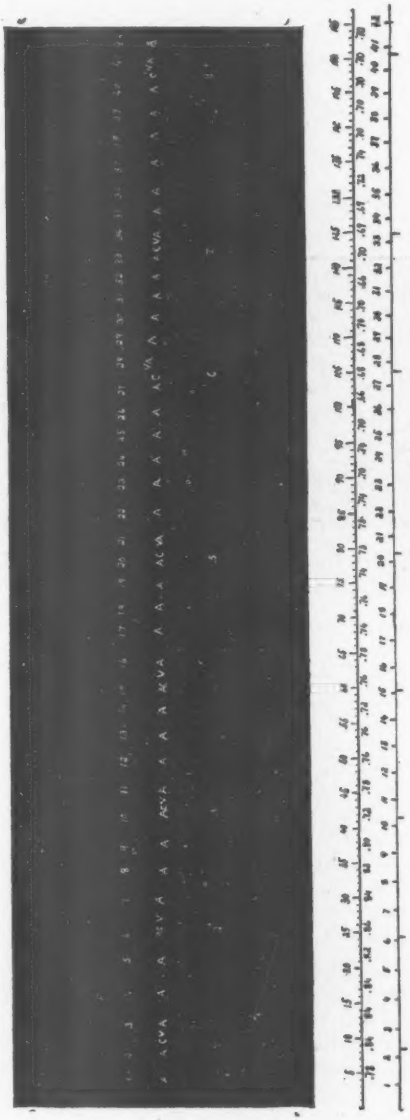


FIG. 3.—February 8, 1910. Before atropine. Complete block. Irregular ventricular action.

artery, we should here in this case have a *c* wave, because, judging from the radial, the left ventricle contracted with its customary strength and at the proper time. Therefore, it seems to us that the *c* wave cannot be due to the carotid, but must be due to the right ventricle, and that in this particular instance the right ventricle did not contract or contracted feebly, although the left did contract normally. Coming as this did just before the institution of the 2 to 1 rhythm, it certainly suggests that the right ventricle ceased first, and then the left ventricle followed suit, and that then a perfect 2 to 1 rhythm resulted. This would seem to prove that one ventricle can contract normally while at the same time the other ventricle either contracts very feebly or not at all. Probably it would be unwise

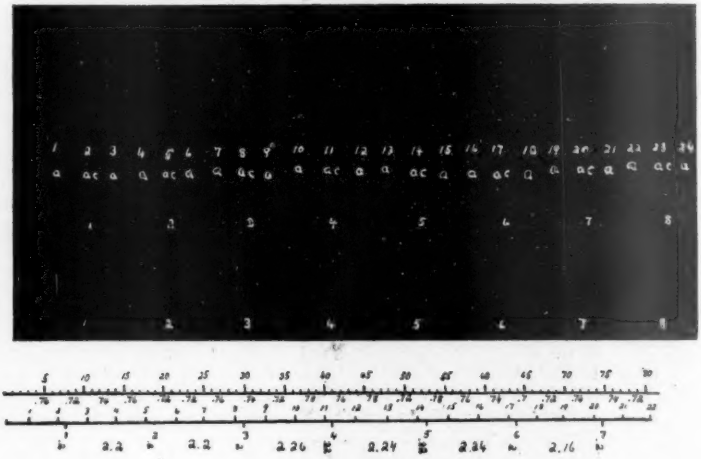


FIG. 4.—February 8, 1910. After atropine had been given. Incomplete block with 3 to 1 rhythm. *a-c* interval usually normal.

from this single observation to say that in this particular case the division of the bundle of His going to the right ventricle was more involved or damaged than the division going to the left ventricle, but if further similar findings had been noted, possibly such a localization could have been made.

The earliest reference to hemisystole we could find was in an article by Charcelay in 1838, who wrote a memoir on some remarkable cases of asynchronous contraction of the ventricles of the heart. Then in 1868 von Leyden[2] reported the first of his series of cases, the last of which appeared in 1908, forty years later. These cases of von Leyden's have been considered by most other writers as

[2] Virchow's Archiv, 1868.

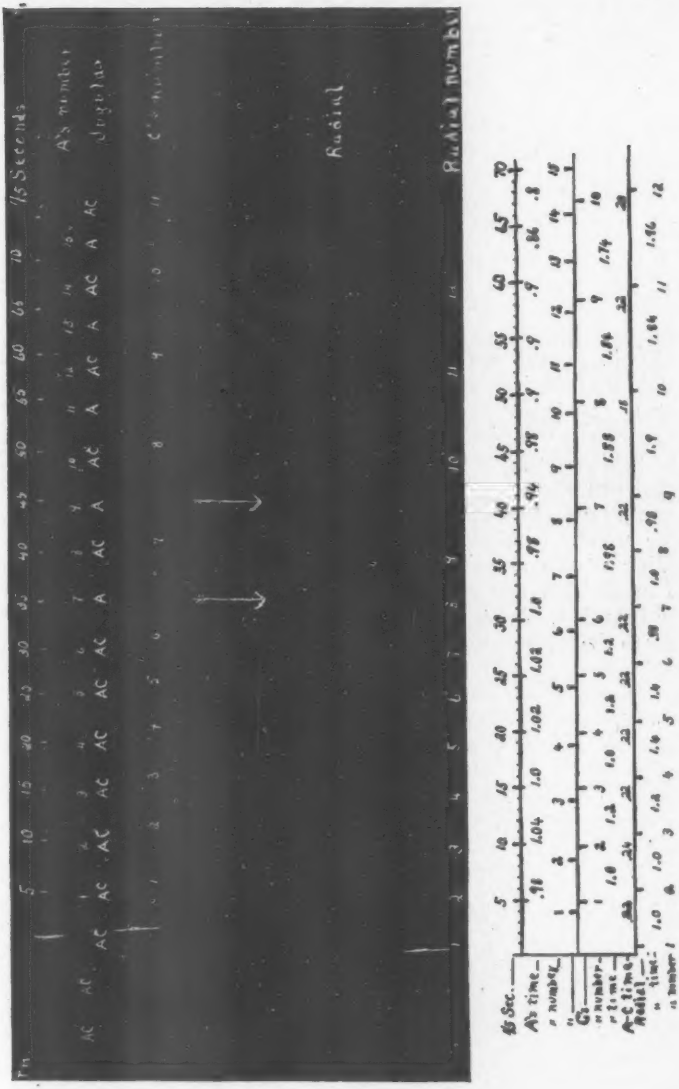


FIG. 5.—February 17, 1910. Change from the normal rhythm to a 2 to 1 rhythm. Note that the seventh *a* wave is *not* followed by a *c* wave although it *is* accompanied by a wave in the radial (No. 8), and that the ninth *a* wave is *not* followed by a *c* wave and is *not* accompanied by a wave in the radial.

typical examples of pulsus bigeminus. Hering, in particular, has been very insistent upon this point, and thinks that the term hemisystole should be abolished, because although on inspecting the hearts of dying animals one can observe one ventricle beating after the other has ceased to beat, or one ventricle contracting more rapidly than its fellow, such contractions as these can only be noted in dying hearts, and are not, he thinks, compatible with life.

Barker and Hirschfelder,[3] in a very important paper entitled "The Effects of Cutting the Branch of the His Bundle going to the Left Ventricle," reported that they found, when they cut the left branch without injuring the right, that complete block resulted, but both ventricles contracted synchronously. In one experiment no block resulted, and they, therefore, conclude that the bundle of His plays little if any role in the coördination of the two ventricles.

Eppinger and Rothberger[4] report some studies of the consequences of section of Tawara's branches of the conducting apparatus. Their observations of the electrocardiograms they obtained showed after section of the left branch a curve significant of right-sided extrasystoles, and on section of the right branch, left-sided extrasystoles. When the ventricular contractions are infrequent, the auricular waves can be seen before the ventricular. The authors conclude from their studies that the stimulus originating in the auricles passes down through the bundle of His to its division and then to the left and right ventricles through the branches. When the left branch is cut, the impulse does not flow to the left ventricle, and it is, therefore, not stimulated to direct contraction; the right ventricle contracts first and the left receives its impulse in an indirect way through the right. This state of affairs is the same as occurs when in case of vagus inhibition the right ventricle is stimulated from its wall and consequently contracts before the left. The authors obtained somewhat similar results in a previous study in which cardiac lesions were produced by injections of silver nitrate solution.[5] They refer to the fact that Aschoff had raised the question in connection with his pathological studies whether a lesion of one of the Tawara branches or its division would not impair cardiac action, and Monckeberg has shown that ecchymotic hemorrhage and subendocardial fatty metamorphosis is apt to occur in the position of the conducting bundle and its branches. The authors finally call attention to the fact that in their operations on dogs, section of one Tawara branch was followed by gallop-rhythm characterized by a disturbance of synchronous contraction of the ventricles. In the Medical Clinic of Vienna, they recently observed two fatal cases in which during life the form of the electrocardiogram had suggested a diagnosis

[3] Trans. Assoc. Amer. Phys., 1909.
[4] Ztsch. f. klin. Med., lxx, 1.
[5] Wien. klin. Woch., 1909, No. 31.

of interruption of conduction to one ventricle. The autopsies showed macroscopically a large myocarditic lesion in the septum. How far this may have invaded the conducting tissues will have to be determined by careful microscopic examination.

Pulsus bigeminus simulates hemisystole because the second or smaller beat, which is an extrasystole, although giving a distinct wave in tracings made from the apex and in the neck does not give any wave in the radial. McKenzie has always insisted that the *c* wave was caused by the carotid, and this view has been adopted by some and rejected by others. It is our belief that the *c* wave can be caused by the carotid artery if the recorder be placed directly over this vessel, but we also believe that by moving the recorder a venous wave can be obtained that has nothing to do with the carotid but comes from the right ventricle. In other positions both these factors undoubtedly may take part in the formation of the so-called *c* wave.

6. A study of a number of the tracings showed that when the rhythm was normal the *a–c* interval averaged 0.2208 second, this figure being taken from 60 measurements; when the rhythm was 2 to 1 the *a–c* interval averaged 0.205 second, as estimated from 24 measurements; when the rhythm was 3 to 1 the same figure was obtained from the average of 7 measurements. Greater variations were noticed in the length of the *a–c* interval during normal rhythm than at other times. No evidence of progressive increase in the length of the *a–c* interval leading up to a partial block was found at any time.

7. That the auricular rate is slower when normal rhythm exists and faster when a 2 to 1 rhythm has come on, is found to be true from a series of measurements taken from a number of tracings. These changes in rate come on immediately after the change one way or other sets in, but we find that there is a greater immediate disturbance of the auricular rate of contraction when the rhythm changes from a 2 to 1 to a normal rhythm than when the normal changes to a 2 to 1 rhythm. Thus, we find an average drop of some 17.9 beats per minute in the rate or a change from 74.2 to 56.3 when there was a restoration to normal rhythm; while when the normal rhythm changed to a 2 to 1 rhythm there was an increase in the auricular rate of but 10.6 beats in the minute or a change from 59.7 to 70.3. These averages were obtained by measuring only about 20 beats immediately following the change in rhythm.

8. In attempting to determine the cause of greater auricular rate during 2 to 1 or 3 to 1 rhythm than when no dissociation existed, it has occurred to us as reasonable that the interference with auricular outflow, which is present at each alternate or third auricular contraction, would necessarily leave the auricle relatively unemptied and would thus occasion an earlier stimulus to contraction, which would result in a more rapid rate. When a change from normal to

2 to 1 rhythm occurs the quickening of auricular rate is less prompt than is the slowing when a 2 to 1 rhythm changes to the normal. An explanation for this circumstance may be found in the fact that resumption of normal rhythm after a 2 to 1 rhythm at once relieves the auricle of back pressure and the consequent stimulus to increased rate. Such a sudden relief is followed by prompt re-adjustment of auricular activity, whereas the effect of change from normal to a 2 to 1 rhythm and the consequent gradual overfilling of ventricle and auricle would be met by a slower response.

In this case during the time of complete block the ventricular rhythm was not at all times regular, as has been already mentioned, although the ventricle usually contracted about 20 times to the minute, or about every three seconds; at other times there were irregular pauses, the longest being 5.4 seconds. There were, however, no extrasystoles at any time. It is known from physiological experiment that, owing to poorly developed or retained independent rhythmicity, a portion of the ventricle, as, for example, the severed apex of the ventricle, tends to cease contracting after a time and will resume regular contractions only after an external stimulation. It is, therefore, possible that the continued action of the ventricle sometimes for long periods and despite occasional long pauses in cases of complete block may be due to the stimulus of distention with blood resulting from the continued auricular action.

THE TREATMENT OF ANEURYSM OF THE AORTA BY THE INTRODUCTION OF WIRE AND THE PASSAGE OF A GALVANIC CURRENT.[1]

By Augustus A. Eshner, M.D.,

PROFESSOR OF CLINICAL MEDICINE IN THE PHILADELPHIA POLYCLINIC AND COLLEGE FOR GRADUATES IN MEDICINE; PHYSICIAN TO THE PHILADELPHIA GENERAL HOSPITAL; ASSISTANT PHYSICIAN TO THE PHILADELPHIA ORTHOPEDIC HOSPITAL AND INFIRMARY FOR NERVOUS DISEASES; CONSULTING PHYSICIAN TO THE MERCY HOSPITAL, PHILADELPHIA.

Aneurysm of the aorta is essentially a fatal disorder sooner or later, partly on account of the resulting interference with the blood distribution and pressure phenomena, but largely and inherently because of the underlying degenerative state of the vessel wall, so that if correction or repair be effected in one situation, active manifestations are likely to appear in another situation. The affection is almost invariably associated with more or less widespread atheroma of the arterial system, and antecedent syphilis appears to be far and away the commonest cause. The dilatation or diverticu-

[1] Read at a meeting of the Association of American Physicians, Washington, D. C., May 4, 1910.

lation of the bloodvessel must, accordingly, be looked upon merely as a local expression of changes widely distributed throughout the vascular system, and accordingly the results of any therapeutic procedure can at best be only palliative, except in the small group of cases in which the lesion arises from purely local influences, such as traumatism or adjacent disease. Nevertheless, it will not be gainsaid that measures of temporary relief are often indicated, either for the amelioration of symptoms or for the prolongation of life. In this connection the fact should not be ignored that under certain conditions life may be continued for many years in the presence of an aneurysm and also that a sacculated aneurysm of the aorta may, and occasionally does, undergo spontaneous cure by the total occlusion of the aneurysmal sac by fibrous laminated adherent clot, and be discovered at autopsy only as an incidental phenomenon. Furthermore, it is impossible to foresee the outcome of the brilliant experimental work that has been done and is being continued in the plastic surgery of the vascular system. Nevertheless, there will remain cases in which symptomatic treatment only will be possible.

In the treatment of aneurysm of peripheral vessels, with abundant anastomotic connections, deligation, with or without excision or resection of the aneurysmal sac, has yielded satisfactory and at times even brilliant results, but this mode of procedure is scarcely applicable to aneurysm of the aorta, and more particularly, its thoracic division, partly on account of anatomical limitations with respect to the establishment of a collateral circulation, and partly by reason of the practical inaccessibility of the disease focus. Various modes of treatment, dietetic, medicinal, physical, have been applied to aneurysms of the latter class, with varying measure of success. Among the most rational and most useful of these may be mentioned rest, restricted diet, ergot, iodides, calcium chloride, adrenaline, introduction of foreign bodies, electricity. Of foreign bodies there have been introduced into the aneurysmal sac substances such as horsehair, catgut, silk, watchspring, needles, and wire of various kind.

Charles H. Moore and Charles Murchison[2] first employed fine iron wire in this manner, introducing twenty-six yards through a cannula into an aneurysm of the thoracic aorta. The tumor was at first reduced in size, but subsequently it became larger, and the patient died on the fifth day. Postmortem examination disclosed abscess of the left kidney and pericarditis.

The possibility of treating aneurysm by transfixion with needles occurred to Velpeau[3] and to Benjamin Phillips;[4] while Ciniselli[5]

[2] Medico-Chirurgical Transactions, 1864, xlvii, 129.

[3] Memoires de l'Academie des Sciences, December, 1830.

Medical Gazette, 1831, 499; also pamphlet, A Series of Experiments for the Purpose of Showing that Arteries may be Obliterated without Ligature, Compression, or the Knife, 1832.

[4] Gazette des Hôpitaux, 1868.

recommended galvanopuncture or electrolysis by means of needles introduced into the aneurysmal sac.

Burresi and Corradi[6] introduced 42 cm. of No. 30 annealed wire through a cannulated needle into the sac of a large aneurysm of the ascending division of the arch of the aorta, and passed a constant current. Temporary improvement ensued, but death took place three and a quarter months after the operation. This latter procedure variously modified has since been carried out in a moderate number of cases—of which I have been able to tabulate 38—and the results appear to justify its employment under suitable conditions. It is applicable only to sacculated and not to fusiform aneurysms, particularly if lesser measures have failed, and even and perhaps especially when alarming symptoms are present or rupture is threatened. The most that can be hoped for under such circumstances is amelioration of symptoms and prolongation of life, and these ends have been attained in most instances in which the procedure has been employed. In isolated instances the inevitable fatal issue appears to have been hastened, but in the vast majority the intervention has been justified by the results.

The technique of the procedure is extremely simple. Under aseptic conditions a hollow needle of gold, silver, or platinum, insulated except at its tip, is introduced to a considerable distance into the most prominent part of the aneurysmal sac, care being taken to avoid puncture of the remote opposite side of the vessel-wall. Through the needle gold or silver wire is fed in amount sufficient loosely to fill the sac. Ordinarily ten or twelve feet of wire will be sufficient. Should the wire kink before the desired amount is introduced, the needle may be withdrawn and introduced in another situation, or a second needle may be introduced, and wire insinuated through it. Indeed, several needles may be introduced at the same sitting. The end or ends of wire are connected with the positive pole of a galvanic battery and a current gradually increased to 60, 80, or 100 milliampéres and as gradually decreased, is passed over a period of from an hour to an hour and a half. The negative pole of the battery is placed upon some indifferent point, as upon the back, A spurt of blood usually follows introduction of the needle, but the hemorrhage is not considerable and it soon ceases. Pulsation may begin to subside during the introduction of the wire, but, as a rule, this effect does not appear until the passage of the current, and at the same time the patient often expresses relief from distressing symptoms, such as pain and dyspnœa. In many instances the improvement is progressive and persists for a varying period of time, and in one case it was maintained for nearly twelve years. The operation is remarkably free from accident or complication, transient hemiplegia having been exceptionally observed. Death

[6] Lo Sperimentale, April, 1879, p. 445.

may result from hemorrhage or the effects of pressure and exhaustion, or from rupture of a second aneurysm, and at times from an entirely unrelated cause. Only sacculated and not fusiform aneurysms are suitable for the operation. Experience and experiment have shown that gold or silver wire is the best to employ, the latter having the advantage of lesser cost. The positive pole is preferred for connection with the wire on account of the firmer clot produced at this pole.

In addition to the recorded cases, I am able to report two that have recently been under my observation.

CASE I.—A sheetmetal worker, aged sixty-four years, was admitted to the Philadelphia General Hospital March 26, 1909, complaining of dyspnea, pain on both sides of the chest, cough, expectoration, headache. The dyspnea has been present for a year, and had become worse six weeks previously. At this time the pain in the chest appeared, and a cough of long standing became aggravated. The man had worked at his trade until four weeks before admission to the hospital, when he was compelled to give up on account of the difficulty in breathing. Physical examination disclosed the signs of effusion into the left pleural cavity, and on aspiration about three pints of clear serous fluid were evacuated. The apex-beat of the heart, which previously had been one and a half inches to the right of the sternum, receded to the left of the sternum. The normal heart sounds were replaced by murmurs, but little transmitted. The voice was hoarse, and there was a metallic cough. Bulging, with pulsation, was visible on the upper anterior aspect of the left chest, with dulness on percussion and a to-and-fro murmur on auscultation. Tracheal tugging was distinct. The percussion note over the greater part of the left chest was dull, with diminished vocal resonance and fremitus, and friction sounds toward the base. Over both lungs bubbling rales were audible. On laryngoscopic examination the left vocal band was found immobile. The urine was free from albumin and sugar. *X*-ray examination disclosed an aneurysm of the aorta.

As the swelling and the pulsation of the left chest increased and the general symptoms grew worse the proposition was made to the patient that wire be introduced into the sac and a galvanic current be passed, and in this he acquiesced. Accordingly, on April 24, 1909, after injecting $\frac{1}{4}$ grain of morphine hypodermically, and inducing local anesthesia with cocaine, Dr. Ralph S. Lavenson introduced at the most prominent point an insulated hollow gold needle in the second interspace just to the left of the sternum. A small amount of dark, thick blood oozed from the needle. About twelve feet of gold wire was introduced into the sac, blood meanwhile flowing more freely. The end of the wire was connected with the positive pole of a galvanic battery, while a large wet pad connected with the

negative pole was applied to the back. The current was started at 5 milliampéres and thus continued for three minutes, being increased 5 milliampéres every succeeding three minutes until a total of 60 milliampéres was reached. It was maintained at this level for ten minutes, and then gradually reduced 10 milliampéres every three minutes. Fifty-eight minutes were consumed in the passage. During its progress pulsation diminished. The patient withstood the operation well and made no complaint of pain. Subsequently there was temporary return of the pain on both sides of the chest. For a time also a tender swelling made its appearance at the site of puncture, but this soon subsided. The patient, however, failed to gather strength, and troublesome cough persisted, and without the development of other symptoms death took place from exhaustion on May 22, 1909, four weeks after the operation.

There was some delay in securing an autopsy, and this was not held until five days after death. The body has been embalmed and was in a good state of preservation. The right lung was bound by moderate adhesions, which were readily detached. The left lung was held by more numerous and firmer adhesions, and it was greatly compressed. The anterior mediastinum was occupied by the heart and a large, firm mass. The heart was in a somewhat transverse position, the right auricle being situated to the right and the right ventricle to the left, while the left auricle and ventricle were altogether posterior. The pericardial cavity was obliterated by adhesions. The ascending and transverse portions of the arch of the aorta were the seat of an immense aneurysm as large as a child's head, and almost entirely filled with clot which in places had undergone fibrous transformation, and in the midst of which was a coil of gold wire. The descending portion of the arch and the thoracic aorta were the seat of marked atheroma, with calcareous deposits. The liver was large, hard, and cirrhotic. The kidneys were large and cyanotic, with parenchymatous and interstitial changes. The spleen was soft and voluminous. The lungs were firm and on section frothy.

Case II.—V. M., an Italian laborer, aged fifty-nine years, was admitted to the Philadelphia General Hospital, September 10, 1908, complaining of precordial pain, cough, expectoration, dyspnœa, and weakness. He had been a saloonkeeper for thirty-two years and had indulged in alcohol to excess. He had suffered from several of the diseases of childhood, from smallpox at six, and he had had an attack of gonorrhœa at twenty, and a chancre at thirty. For eight months he had been complaining of pain in the precordium, progressive in severity, radiating in the course of the great vessels and extending down the left arm, together with cough and expectoration. The pulse was small, slow, and thready, the right smaller and slower than the left. Pulsation was visible in the vessels of the neck. The chest

was barrel-shaped and presented a pulsating swelling to the left of the sternum between the second and fourth intercostal spaces, and yielding dulness on percussion. No thrill was palpable, but a bruit was audible on auscultation. The action of the heart was deliberate, rhythmic, and forcible; the first sound was clear, the second accentuated at the base. The pupils were moderately dilated, but they reacted to light and in accommodation. The urine was free from albumin and sugar. Skiagraphic examination disclosed an aneurysm of the arch of the aorta. On September 28, fourteen feet of gold wire was introduced by Dr. Ralph S. Lavenson into the pulsating mass, and a galvanic current was passed. The current strength was begun at 5 milliampéres and was gradually increased to 75 milliampéres and as gradually reduced. The patient was somewhat restless during the course of the procedure, and he coughed slightly with each increase in the strength of the current. Relief was afforded by a hypodermic injection of morphine, $\frac{1}{4}$ grain, and the patient slept for a portion of the time. Pulsation soon diminished and became almost imperceptible. Pain, however, persisted, and at a later date pulsation returned. On May 1, 1909, the patient went to visit friends, and he returned on May 5 with a pulsating mass of increased size at the site of the earlier swelling. Death took place suddenly on May 6, seven and one-fourth months after the operation.

On postmortem examination a globular swelling was visible upon the anterior aspect of the left side of the chest between the second and fifth ribs and the median and nipple lines, and the centre of which exhibited a dark-red discoloration. The underlying third rib was fractured. The right pleural cavity was free and the right lung was covered at its apex by a small area of thickened pleura, but it was in other respects normal. The left pleural cavity contained 200 c.c. of slightly cloudy, straw-colored fluid, in which floated flakes of fibrin. The pleural surfaces were moist and glistening. The left lung was pale gray in color and crepitant throughout. On section a considerable amount of frothy fluid escaped. The pericardial cavity contained a large amount of currant-jelly clot under such pressure that it was forcibly extruded when the pericardium was incised. The heart was flabby and covered by a considerable amount of epicardial fat. The myocardium was pale and cloudy. All of the chambers of the heart were dilated, but the walls were not hypertrophied. The valves and orifices presented no abnormality. The aorta was extremely atheromatous, the intima in places being punctured and fissured. The ascending and transverse portions of the arch of the aorta were greatly dilated and from the anterior aspect of the latter bulged a large sacculated aneurysm measuring 8 x 6 cm. The sac was largely filled by organized thrombus and recent blood clot, in the midst of which,

loosely coiled, lay 350 cm. of fine gold wire. The overlying third rib was partially eroded and fractured, and the aneurysm was adherent to the second and fourth ribs. A short distance above the aortic cusps the vessel wall was softened and eroded, and it was at this point that the fatal hemorrhage into the pericardium had taken place. Several other weakened spots in the wall with beginning bulging were visible in other parts of the aorta. The spleen was considerably enlarged. It was friable and it tore in removal. Its structure was pale, the capsule presenting a variegated appearance, with dark and red spots. The kidneys were pale and flabby, the capsule stripping easily and leaving a smooth surface behind. On section they presented a cloudy appearance, the cortex being slightly increased in thickness and pale in color. The right kidney was smaller than the left. The liver was enlarged and measured 20 x 18 x 9 cm. It was pale and flabby and its capsule was smooth. Section presented a dirty yellow appearance; the acini were not visible.

The accompanying table comprises 34 cases that I have been able to collect from the literature, together with 2 cases recently reported by Beardsley, Rhoads, and Saurman at a meeting of the College of Physicians of Philadelphia, February 3, 1910, and the two cases of my own.

In addition Hunner[7] refers to a case of Dr. John Neff, of Baltimore, in which operation was performed on three occasions by D. D. Stewart, of Philadelphia, and apparently not otherwise reported. The patient was a travelling salesman, with a large thoracic aneurysm; ten feet of wire was introduced February 5, ten feet April 1, and five feet June 5. Death took place from rupture and hemorrhage November 17.

Dr. Robert N. Wilson, Jr., of Philadelphia, informs me that he has treated 3 cases of aneurysm of the ascending portion of the arch of the aorta and 1 case of aneurysm of the descending portion by the introduction of 15 feet of gold wire and the passage, throughout an hour, of a galvanic current gradually increased to 160 milliampéres and then gradually diminished. In three of the patients death appeared imminent, and although they were helped symptomatically, life was prolonged but a few days. One had been wired twice previously without advantage, and the patient was able to return to work after the third operation, and was still living at the date of Dr. Wilson's letter to me.

Of the 38 cases included in the table that I have prepared, 36 occurred in males, 1 in a female, and in 1 the sex was not mentioned. The age of the patient was mentioned in 35: Five were between twenty-five and twenty-nine, 3 between thirty and thirty-four, 6 between

[7] Loc. cit.

thirty-five and thirty-nine, 7 between forty and forty-four, 4 between forty-five and forty-nine, 4 between fifty and fifty-four, 4 between fifty-five and fifty-nine, 1 was sixty-four, and 1 was seventy-three. The arch of the aorta was the seat of the aneurysm in 18 cases (ascending portion in 9, descending portion in 3, "arch" in 2, transverse portion in 2, ascending and transverse portions in 1, transverse and descending portions in 1), the thoracic aorta in 5, the abdominal aorta in 5, the innominate artery in 3, the subclavian artery in 2, the superior mesenteric artery in 2, the thoracic and abdominal aorta in 1, the splenic artery in 1, and the cœliac axis in 1. Gold wire was used in 16 of the cases, silver wire in 15, steel wire in 4, and silver-plated copper wire in 1. The duration of life following the operation was as follows: Five and one-half hours in 1, twenty-four hours in 1, forty hours in 1, forty-two hours in 1, two days in 1, five days in 1, six days in 1, seven days in 1, nine days in 1, eighteen days in 1, twenty days in 2, three weeks in 1, twenty-three days in 1, three and one-half weeks in 1, four weeks in 1, five weeks in 1, forty-seven days in 1, two months in 1, eleven weeks in 1, three and one-fourth months in 1, four months in 1, five months in 1, six months in 2, seven months in 1, seven and one-fourth months in 1, eight months in 2, eight and one-half months in 1, ten months in 1, fourteen months in 1, three and one-half years in 1, five years in 1, eleven years and eight months in 1. One patient was living three years after the operation, but his subsequent history is unknown (Kerr, loc. cit.).

Conclusions. Aneurysm of the aorta is essentially a fatal disorder, although exceptionally spontaneous cure takes place. Accordingly, treatment can, as a rule, be only palliative, relieving symptoms and prolonging life. Deligation is inapplicable from anatomical considerations. Rest, ergot, iodides, gelatin, calcium chloride, adrenalin chloride and introduction of foreign substances, with or without the passage of a galvanic current, have been employed. Horsehair, catgut, needles, watchspring, and wire of various kind have been introduced, with varying result, and a galvanic current has been passed through conducting substances. Wiring with electrolysis has been employed in a moderate number of instances, with satisfactory results in the majority. The procedure has proved simple and safe, but it is applicable only to sacculated and not to fusiform aneurysms.

Reporter.	Sex.	Age.	Situation.	Operation.	Result.	Remarks.
Burresi-Corradi, Lo Sperimentale, April, 1879, 445	M	43	Ascending arch	42 centimeters No. 30 annealed wire; 16 cells, 25 minutes	Death, three and one-quarter months	
Richard Barwell, Lancet, June 5, 1886, 1058	M	39	Descending arch	10 feet finest steel wire; 9 to 10 milliampères, 70 minutes	Death, seven days	Exhaustion; rupture of second sac
J. W. Roosevelt, Medical News, 1887, 397	M	25	Arch	225 feet fine steel wire (oo); 25 milliampères, 30 minutes	Death, twenty-third day	Gangrene toe
Robert Abbe, Medical News, 1897	M	46	Subclavian	100 feet No. 1 catgut; 8 days later 150 feet fine steel wire; 50 to 100 milliampères, 70 minutes	Death, second day	Current reversed; dissecting; rupture into trachea
W. W. Kerr, Occidental Medical Times, January, 1889, 1	M	38	Ascending arch	6 feet drawn silver wire; 50 minutes	Death, eighteenth day	Fusiform
W. W. Kerr, Occidental Medical Times, January, 1889, 1	M	56	Ascending arch	Galvano-puncture; later 10 feet wire, 30 minutes	Working as a pavior three years after operation*	Fusiform
Julius Rosenstirn, Amer. Jour. Med. Sci., 1891, ci, 55	M	25	Ascending arch	Galvano-puncture; later 26 feet silver wire, 30 minutes	Death, eleven years and eight months*	
D. D. Stewart, Trans. College of Physicians of Philadelphia, 1892, xiv, 68	M	30	Thoracic and abdominal	Heavy silver wire; 70 milliamperes, 1 hour	Death, ninth day	Rupture
E. P. Hershey, Therap. Gaz., 1896, 590	M		Innominate	2½ feet No. 28 gold wire; 70 milliampères, 45 minutes	Death, fourteen months	
D. D. Stewart and J. L. Salinger, Amer. Jour. Med. Sci., 1896, cxii, 171				10 feet No. 30 gold wire; 30 to 80 milliampères, 75 minutes	Death, three and one-half years	Death from cerebral thrombosis
D. D. Stewart, Trans. College of Physicians of Philadelphia, 1897, xix, 38	M	40	Innominate			
W. H. Noble, Phila. Med. Journal, June 25, 1898, 1203	M	37	Abdominal	8½ feet No. 30 gold wire; 70 milliampères, 37 minutes	Death, eight months	Abdominal section; death not due to aneurysm
H. A. Hare, Therap. Gaz., 1898, 293; 1900, 9; Penna. Med. Journal, 1898-1899, 312	M	46	Transverse and descending arch	9 feet fine gold wire, 80 minutes	Death, seven months	Death from exhaustion; second aneurysm distal
D. D. Stewart, Phila. Med. Journal, Nov. 12, 1898, 1011	M	40	Splenic	10 feet silver wire; 50 milliampères, 30 minutes	Death, fifth day	Abdominal section
D. D. Stewart, Phila. Med. Journal, Nov. 12, 1898, 1011	M	42	Thoracic	14 feet fine gold wire; up to 80 milliampères, 80 minutes	Death, fifth day	Life prolonged; comfort afforded
J. C. Reeve, Annals of Surgery, 1899, xxx, 704	M	49	Abdominal	7 feet silverplated, soft copper wire; 80 milliampères, 50 minutes	Death, twenty-four hours	Abdominal section; strand of wire extended to aortic valves
E. S. Corson, Phila. Med. Journal, March 4, 1899, 511	M	31	Ascending arch	6 feet drawn silver wire; 8 dry cells. 2 hours	Death, forty-two hours	Death from arrest of respiration
H. A. Hare, Therap. Gazette, 1900, 9	M	48	Arch	10 feet gold wire; 1 month later 10 feet gold wire in each of two places; 10 to 100 milliampères, 90 minutes	Death, eight months	Life prolonged
H. A. Hare, Therap. Gazette, 1900, 510				10 feet gold wire; 5 days later 10 feet gold wire; up to 85 milliampères, 1 hour	Death, sixth day	Death from respiratory failure
G. L. Hunner, Bulletin Johns Hopkins Hospital, Nov. 1900, 263	M	27	Abdominal	No. 27 undrawn silver wire; kinked; withdrawn; 12 minutes; second wire, 17 inches; third wire, 38 inches; 30 to 100 milliampères, 85 minutes	Death, forty hours	Abdominal section; symptoms of internal hemorrhage; death probably hastened

G. L. Hunner, Bulletin Johns Hopkins Hospital, Nov. 1900, 263	M	25	Abdominal superior mesenteric	5 feet silver wire (copper alloy); 30 to 70 milliampères, 80 minutes	Death, twentieth day	Abdominal section; death from hemorrhage
G. L. Hunner, Bulletin Johns Hopkins Hospital, Nov. 1900, 263	M	51	Thoracic	10 feet silver wire (copper alloy); 10 to 20 milliampères, 75 minutes	Death, six months	
G. L. Hunner, Bulletin Johns Hopkins Hospital, Nov. 1900, 263	M	39	Thoracic	10 feet silver (copper) wire; 10 milliampères, 1 hour; after 24 days, 10 feet silver (copper) wire; 10 milliampères, 1 hour	Death, eleven weeks	Death from compression of trachea
G. L. Hunner, Bulletin Johns Hopkins Hospital, Nov. 1900, 263	M	30	Abdominal	8 to 9 feet higdly drawn sterling silver wire; 10 milliampères, 1 hour	Death, fifth year	Fusiform aneurysm; abdominal section
H A. Hare, Therapeutic Gas., 1903, 19	M		Thoracic	9 feet gold wire; second operation 6 months later	Death shortly,	Repeated copious hemorrhage
J. Daland, Penna. Med. Journal, 1903-1904, 134	M	52	Left subclavian	20 feet gold wire; 1 to 80 milliampères, 110 minutes	Death, twentieth day	Thrombosis of carotid artery from pressure of aneurysm
Raymond Russ, Occidental Medical Times, 1903, 284	M	50	Transverse arch	1 foot No. 26 soft silver wire; 50 to 100 milliampères, 55 minutes; 15 days later, 6 feet No. 22 silver wire; 50 to 60 milliampères, 40 minutes	Death, five weeks	Pressure on trachea
J. L. Salinger, Therapeutic Gazette, 1903, 438	M	73	Ascending arch	16 feet wire; 5 to 65 milliampères, 75 minutes	Death, five months	Transitory hemiplegia and aphasia on third day; death from pneumonia
R. C. B. Maunsell, British Med. Journal, June 18, 1904, 1421	M	28	Abdominal (celiac axis)	6 yards No. 28 silver wire; 1 to 65 milliampères, 30 minutes	Death, forty-seven days	Abdominal section; hemorrhage
H. A. Ballance, Lancet, Oct. 1, 1904, 939	M	44	Ascending arch	8 feet No. 9 silver wire; 24 to 42 milliampères, 45 minutes	Death, ten months	Great relief of symptoms; old emboli kidneys; recent infarct lung
H. A. Hare, Therapeutic Gazette, July, 1905, 433	F	50	Transverse arch	8 feet gold wire; 5 to 50 milliampères, 30 minutes	Death, six months	
H. A. Hare, Therapeutic Gazette, July, 1905, 433	M	42	Innominate	2 feet gold wire; 5 to 30 milliampères, 40 minutes	Death, two months	Exhaustion; interference with respiration and deglutition; no trace of wire at autopsy
H. A. Hare, Therapeutic Gazette, July, 1905, 433			Thoracic	9 feet gold wire; 5 to 50 milliampères, 45 minutes	Death, four months	Pressure; exhaustion
Gaston Torrance, Alabama Med. Journal, 1905-6, xviii, 487	M	40	Descending arch	5 to 6 feet No. 27 drawn silver wire; 10 to 20 milliampères, 1 hour	Death, three and one-half weeks	Hemorrhage; rupture into œsophagus
S. A. Griffiths, Lancet, Aug. 12, 1905, 442	M	37	Abdominal	1 foot fine silver wire; kinked; more wire (6 feet); 15 to 25 milliampères, 15 minutes	Death, five and one-half hours	Abdominal section; double loop of wire had passed into aorta two inches; wire connected with negative pole
E. J. G. Beardsley, Samuel Rhoads, J. Shelley Saurman[7]	M	57	Descending arch	32 feet No. 26 gold wire; 5 to 60 milliampères, gradually increased and decreased	Comfortable after four months	
E. J. G. Beardsley, Samuel Rhoads, J. Shelley Saurman[7]	M	37	Ascending arch	18 feet No. 26 gold wire; 5 to 60 milliampères, gradually increased and decreased	Death, three weeks	Death from rupture of aneurysm; fusiform
A. A. Eshner	M	64	Ascending and transverse arch	12 feet gold wire; after 8 weeks 10 feet gold wire; 5 to 50 milliampères, 58 minutes	Death, four weeks	Death from exhaustion
A. A. Eshner	M	59	Ascending arch	14 feet gold wire; 5 to 75 milliampères	Death, seven and one-quarter months	Rupture into pericardium

[7] Personal communication.

ORTHODIAGRAPHY IN PATHOLOGICAL CONDITIONS OF THE HEART AND AORTA[1].

By Thomas A. Claytor, M.D.,
PHYSICIAN TO THE GARFIELD MEMORIAL HOSPITAL AND THE TUBERCULOSIS HOSPITAL,
AND
Walter H. Merrill, B.L., M.D.,
RADIOGRAPHER TO THE GARFIELD MEMORIAL HOSPITAL, WASHINGTON, D. C.

It is scarcely necessary to say that any instrument or method of examination which renders the science of medicine more exact, or which enables us to make more complete and accurate records of pathological findings, should be appreciated by the profession, even though the field of usefulness be comparatively small.

The orthodiagraph is an instrument which enables us to outline in its exact size any organ or object of which the Röntgen rays will cast a sufficient shadow. This result is obtained by carrying a small pencil of the ray through a series of parallel positions about the borders of the object under observation, and recording the results upon a sheet of paper placed behind the subject.[2] By fixing at the same time landmarks such as the outline of the body, the ribs, the diaphragm, etc., the orientation of the object or organ may be made exact.

Accuracy of Results. We speak of outlining the object under observation in its exact size, but such a statement requires some modification. The heart and aorta are in constant motion, expanding and contracting, and it therefore becomes necessary to choose which part of the cycle is to be pictured. We have chosen expansion because it allows of greater accuracy, it being easier to mark the shadow of the heart or aorta as it makes its widest excursion into the light area of the lungs. If the heart is pulsating very rapidly it becomes much more difficult to make a correct diagram because of the fleeting character of the shadow cast. The condition of the lungs is also very important. If they contain dark areas in the neighborhood of the great vessels it may be impossible to obtain satisfactory results. Marked œdema due to cardiac weakness may also render it impossible to outline the heart.

The Practical Value of Orthodiagraphy. A year ago we[3] reported a series of tables compiled from orthodiagrams of the hearts of about one hundred normal men and women, showing the measurements of the anterior projection of the heart's shadow for

[1] Read at a meeting of the Association of American Physicians, Washington, D. C., May 3 and 4, 1910.

[2] For description of the technique, see Claytor and Merrill, Amer. Jour. Med. Sci., 1909, cxxxviii, 549.

[3] Loc. cit.

various weights (the division according to weight was found to be the most practical method). By reference to these tables it is possible to tell just how much and in what direction a heart shadow under consideration may depart from the average. However, after some experience in making diagrams of the heart, it is seldom necessary to resort to the actual measurement of the resulting figure, as one may see at a glance whether or not there is any material distortion of the outline. It should also be remembered that even in norml individuals of like weight and height there may be considerable difference in the size of the heart.

Possibilities of Error in Orthodiagraphy. There is seldom any difficulty in fixing the landmarks as they may be called. The outline of the neck, shoulders, and body, together with the position of as many of the ribs as are desired, can be marked with the pointer before the current is turned on. The interior of the thoracic cavity is easily marked, if the lungs are clear, and the position of the diaphragm determined at any stage of the respiratory cycle which may be desired. We have found that the median line is more accurate when established by measurements made from the lateral aspects of the interior of the thoracic cavity, as the xiphoid is so often distorted and the umbilicus is a little too low for convenience as a fixed median point.

The right auricular curve is usually clearly seen, but on reaching the right vascular curve there may be some difficulty. If this curve happens to be formed by a dilated ascending aorta its pulsations render it easy of recognition, but if the descending cava is the object to be outlined the absence of motion makes it more difficult to distinguish, as enlarged or dark glands may be projected into this area. The aortic curve, while in some instances casting but a faint shadow, can usually be clearly seen. Below this the pulmonary curve, made by the pulmonary artery as it plunges beneath the arch of the aorta, comes into view. Then the left auricular curve may be seen. In normal cases this curve is often not perceptible, as it is produced by the left auricular appendix, while in instances of dilated left auricle this position of the heart casts the shadow. The left auricular curve is most marked in stenosis of the mitral valve. Below this comes the left ventricular curve, which is usually clearly seen until the apex is reached, when because of its transparency, rapid and wide excursion of movement, and because of its close proximity to the diaphragm in all cases, and to the liver in some cases, it is more difficult to see.

There is sometimes difficulty in distinguishing a dilated aorta from a true aneurysm (Fig. 5), but in most instances aneurysms of the ascending, transverse, and descending aorta are clearly seen. The condition of the lungs in aneurysmal cases is sometimes such as to render it impossible to outline the dilated vessel except in its upper portion.

It has been pointed out by Franz M. Groedel[4] that it is very necessary to bear in mind the remarkable motility of the heart. Thus, diagrams taken of the same subject in the same position during quiet respiration, inflation, and deflation of the lungs, may show a marked difference in the cardiac outline. Fig. 4 illustrates this change. If this fact is lost sight of it is easy to draw false conclusions as to the efficacy of exercises and baths in causing surprisingly sudden and improbable alterations in the area of cardiac dullness. We have shown elsewhere the effect of change of bodily position upon the heart.[5]

Furthermore, it should be remembered that the heart rests upon the diaphragm in close proximity to the stomach and liver, so that distention of the former or enlargement of the latter may cause a deflection of the heart to the left or right, thus giving the impression of enlargement on the side toward which it is deflected.

Aneurysms of the Thoracic Aorta. In the study of aneurysms of the thoracic aorta the orthodiagraph is of real value. With it we are enabled to reproduce diagrammatically the dilated aorta in its exact size, and thus, while making or confirming a diagnosis of the pathological condition by a fluoroscopic examination, we are able to add materially to our historic record of the case. There would seem to be no doubt of the fact that a direct inspection of the heart and great vessels by means of the fluoroscope is of far greater value than an x-ray picture. By direct inspection we can detect the pulsations of the aneurysm and thus may distinguish it, in most cases at least, from solid intrathoracic tumors; whereas, the radiographic picture would show only a shadow which might be either an aneurysm or some form of tumor or glandular enlargement. The oblique illumination is also useful in studying thoracic aneurysms.

Valvular Lesions. It has long been known that certain valvular lesions have a tendency to cause dilation and hypertrophy of the various divisions of the heart, this having been proved not only by the anatomical findings after death but by percussion of the cardiac region during life. In the orthodiagraph we have an excellent instrument for making interesting and more or less valuable records of these changes in the heart's outline. We alluded to this in our previous work, but only very briefly.[6] We have selected for illustration from our collection of diagrams of pathological conditions of the heart a few in which the lesions seemed to be sufficiently clear cut to be of value in this paper. Of course, when a number of valvular lesions coexist there can be nothing distinctive about the cardiogram. While there is no doubt that the different valvular lesions cause the heart to enlarge in a more or less characteristic

[4] Ztsch. f. Balneologie, Klimatologie, und Kurort, Hygiene, 1909–10, ii.

[5] Claytor and Merrill, loc. cit.

[6] Claytor and Merrill, loc. cit.

manner, yet it must not be forgotten that this is not invariably the case. The heart that has been for any prolonged period in the state of lost compensation becomes more or less generally dilated,

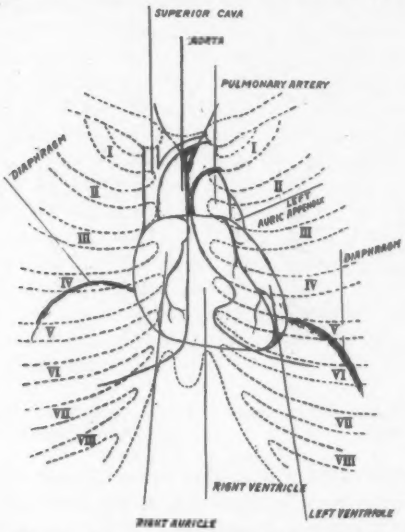


FIG. 1.—The normal heart and great vessels as seen from in front (after Moritz somewhat modified.)

FIG. 2.—Illustration of the method of outlining the heart and landmarks (heart of aortic insufficiency.)

and thus there is likely to be lost any characteristic outline which at an earlier date may have been present.

DESCRIPTION OF ORTHODIAGRAMS. Fig. 1, a somewhat modified diagram after Moritz,[7] shows the normal heart and great vessels as seen from in front.

[7] Strassburg Med. Zeit., August, 1907.

Fig. 2 shows how the neck, shoulders, body, diaphragm and heart are outlined by dots (the heart of aortic insufficiency).

Fig. 3 shows a completed diagram of a normal heart: "right auricular curve" (1); "curve of the great vessels" (2); aortic curve (3);

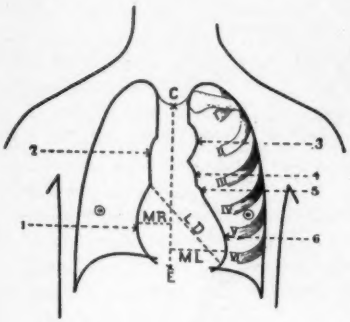


Fig. 3.—Orthodiagram of the normal heart.

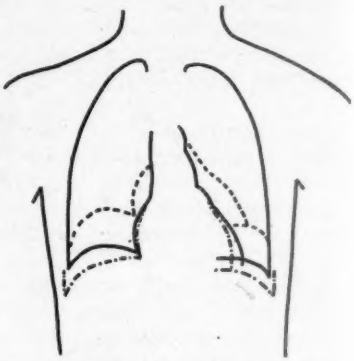


Fig. 4.—Alterations in cardiac outline due to changes in the position of the diaphram.

pulmonary curve (4); left auricular curve (5); left ventricular curve (6); together with the lines upon which the size of the heart is estimated (L. D., longitudinal diameter; T. D., transverse diameter, the sum of M. R. and M. L.)

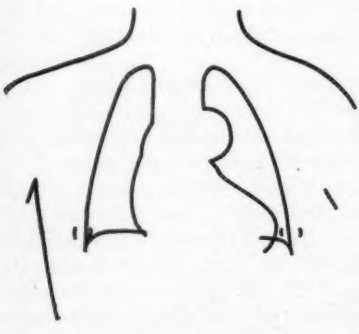


Fig. 5.—Aortic insufficiency—possibly also thoracic aneurysm.

Fig. 6.—Aneurysm of the aorta.

Fig. 4. The continuous line shows the diagram of the heart and diaphragm during quiet respiration. The broken line shows the same during extreme expiration, while the diagram formed by the dots and dashes is taken during deep inspiration. This figure

clearly indicates the remarkable mobility of the heart and, one might say, flexibility of its walls, thus accounting for many mistaken ideas as to the sudden changes in the actual size of the organ as demonstrated by percussion.

Fig. 5 is the heart of a man, aged sixty-six years, who presented the typical signs of an aortic insufficiency. The chief point of interest was as to whether the very marked aortic curve was due to a true aneurysm or only to the dilatation of the arch of the aorta so often seen in aortic insufficiency. Since our notice was called by Dr. F. H. Williams to the remarkably wide excursion of the aortic pulsation in cases of insufficiency of the aortic valve we have frequently noted its occurrence.

Fig. 6 is from a patient with a very large aneurysm of the arch and descending limb of the aorta. Because of the congested and possibly infiltrated condition of the left lung as the result of pressure it was only possible to outline the upper portion of the enlargement. The broken line indicates the probable position of the lower part of the dilatation and of the heart.

FIG. 7.—Diagram of a thoracic aneurysm.

FIG. 8.—The same case three months later.

Figs. 7 and 8 are two diagrams of the same aneurysm, the second having been taken about three months after the first. There had been practically no change, the apparent difference in the size of the heart being due to the fact that the left side of the diaphragm was taken in a lower position in one figure than in the other.

Figs. 9 and 10 are two of our most instructive diagrams, Fig. 9 having been taken sixteen months later than Fig. 10. It may be clearly seen that the aneurysm which is depicted has markedly increased both to the right and to the left. The heart has also enlarged. The prognostic value of the orthodiagraph is here demonstrated. (This patient was seen through the courtesy of Dr. R. W. Baker.)

In a study of changes produced in the heart's silhouette by the various valvular lesions there are several things to be considered:

namely, the age of the patient—the heart in the aged shows a tendency to enlarge and to assume a more oblique position in the chest; the presence of other causes for cardiac hypertrophy, such as arteriosclerosis, chronic nephritis, etc.; the appearance may also

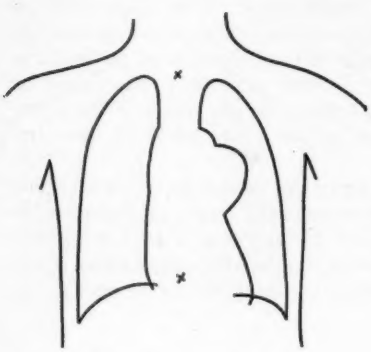


FIG. 9.—Diagram of a thoracic aneurysm.

FIG. 10.—The same case—sixteen months later.

vary depending upon whether the valvular lesion be single, double, or multiple, or whether there has ever been any actual loss of compensation. All of these points, and others not enumerated, are of considerable importance in drawing conclusions concerning a diagram.

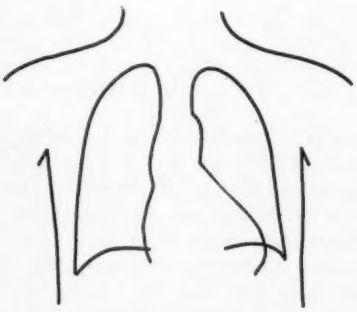


FIG. 11.—Mitral insufficiency.

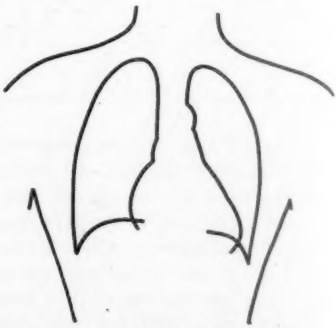


FIG. 12.—Mitral stenosis, well compensated.

Fig. 11 is a diagram of the heart of a man, aged thirty-six years, representing the physical signs of mitral insufficiency with good compensation. The organ is in the vertical position. The pulmonary and left auricular curves, while indistinguishable in this instance,

are enlarged, making the cardiogram rise toward the left axilla. The left ventricular curve is only moderately increased.

Fig. 12 shows the outline of a case of compensated mitral stenosis in a woman, aged thirty-seven years. The chief characteristics are the decidedly vertical position in the chest, the rather full right auricular

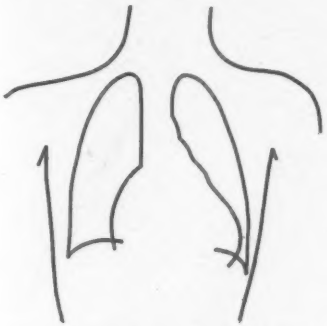


FIG. 13.—Mitral stenosis after prolonged loss of compensation.

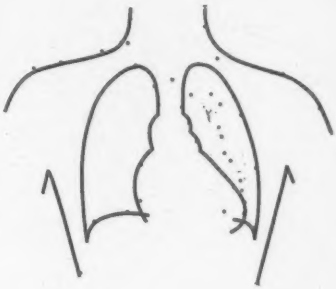


FIG. 14.—Mitral insufficiency and stenosis.

and pulmonary curves, but most striking of all the increase in size of the left auricular curve with the relatively small left ventricular curve, which is just what would be expected because of the fact that there is seldom much increase in the size of the left ventricle in pure mitral stenosis.

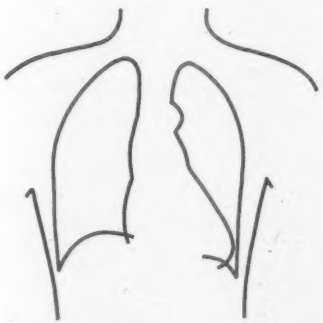


FIG. 15.—Double mitral lesion.

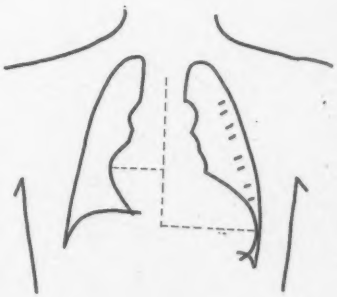


FIG. 16.—Mitral and aortic insufficiency.

Fig. 13 shows the heart of a woman, aged twenty-five years, with a pure mitral stenosis which, however, has passed through a long period of broken compensation. The whole heart is greatly enlarged. The striking features are the bulging right auricular, pulmonary, and left auricular curves.

The outline of the heart with a double lesion may vary, we believe, with the priority of the affection. Thus, in the case of the mitral valve if the insufficiency be the primary trouble the left ventricular curve will be much more pronounced than when the stenosis has been primary.

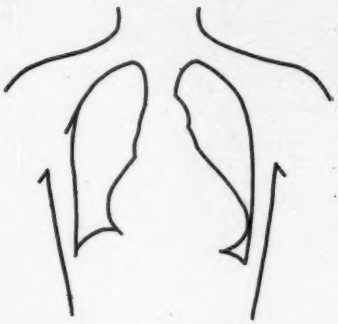


FIG. 17.—Mitral and aortic insufficiency, the mitral probably the more important.

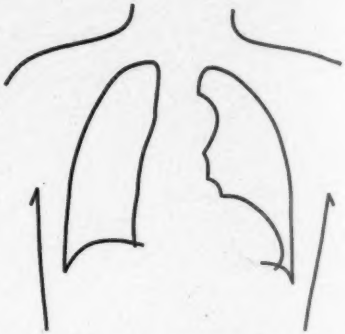


FIG. 18.—Double aortic lesion.

Fig. 14 is of a heart presenting a double mitral lesion in which the stenosis is probably the more important, while in Fig. 15 the insufficency was probably the prior lesion. In both, the right vascular, pulmonary, and left auricular curves are bulging, but in

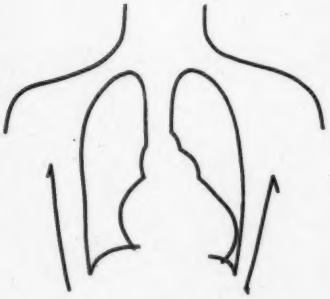


FIG. 19.—Congenital hypoplasia of the aorta.

FIG. 20.—From a case of general arteriosclerosis.

Fig. 15 the left ventricle shows greater hypertrophy. In Fig. 2 we have an excellent example of a pure aortic insufficiency in a man, aged thirty-five years. Both the longitudinal and transverse diameters are considerably increased. The heart is obliquely situated, and is foot-shaped. With this lesion the right vascular and aortic curves

are especially marked because of the dilated condition of the aorta which is so commonly observed. In aortic stenosis the shape of the heart is said to be much the same, but there is no dilatation of the aorta.

Fig. 16 is an example of a combined aortic and mitral insufficiency in a man, aged fifty years. In this instance the aortic was the primary lesion, having preceded the mitral by several years. The hypertrophy and dilatation were enormous, all the curves being greatly exaggerated.

Fig. 17 represents the same lesions in a woman, aged forty years. From the shape of this heart the mitral lesion probably antedated or was more important than the aortic.

Fig. 18 is from a man, aged sixty-six years, with a double aortic murmur. Upon the right side of the heart it may be seen that the right auricular and right vascular curves form a continuous and almost straight line, and this we believe is due to the fact that the pericardium is outlined instead of the heart itself. The heart is of the aortic type.

Fig. 19 is from a young woman who has given evidences from childhood of a moderate degree of cardiac insufficiency. There is a harsh systolic murmur heard over the entire precordium with greatest intensity in the second and third interspaces to the left of the sternum. The heart is rather uniformly enlarged, but the point of interest is the great size of the pulmonary curve, which, as may be seen, is relatively much larger than the aortic. The diagnosis of congenital hypoplasia of the aorta was made some years ago by a very careful observer, and this diagram would appear to support this diagnosis.

Fig. 20 is from a man, aged forty-five years, six feet and one-half inches in height, and weighing 175 pounds. There was a general arteriofibrosis, with high systolic blood pressure, but perfectly normal urine. There were no endocardial murmurs, and the heart would appear to be uniformly enlarged.

It seems unnecessary to multiply diagrams. From those shown it may be seen that the orthodiagraph is of real value to the clinician, and that as it becomes more commonly used we may be able to formulate some useful facts which as yet it is unwise to attempt. The instrument is by no means necessary in all cases, but may be an aid to diagnosis in many more or less obscure pathological conditions of the heart and aorta.

THE ETIOLOGY OF SUBACUTE INFECTIVE ENDOCARDITIS.[1]

By E. Libman, M.D.,
PROFESSOR OF CLINICAL MEDICINE IN COLUMBIA UNIVERSITY; ADJUNCT VISITING PHYSICIAN AND ASSISTANT PATHOLOGIST TO MT. SINAI HOSPITAL,
AND
H. L. Celler, M.D.,
ASSOCIATE IN PATHOLOGY AND ASSISTANT ADJUNCT VISITING PHYSICIAN TO MT. SINAI HOSPITAL, NEW YORK.

During the last eight years we have had the unusual opportunity of making studies in 43 cases of subacute infective endocarditis. Some of these cases were seen in the wards of Mount Sinai Hospital, with the kind permission and coöperation of the attending physicians, Drs. Brill, Koplik, Manges, Meyer, and Rudisch. Many of the cases were seen in the private practice of ourselves or of other physicians who kindly called us in to make bacteriological studies, or to see the cases clinically. In 36 of the 43 cases blood cultures were made during life. Of the 43 cases, 29 were seen since 1906; this shows that the disease is now being more frequently recognized. In many of the cases we were enabled to make not only bacteriological but also clinical, therapeutic, and pathological studies. The details of these investigations, together with a discussion of the literature of the disease and the question of terminology, we shall reserve for a subsequent comprehensive presentation.

The duration of our cases varied from four months to a year or a year and a half. The longer cases may be grouped as the chronic type. Although the cases differed from one another in certain respects, the picture as a whole is a characteristic one, resembling that described by other writers on the disease.[2] In nearly all the cases the disease began insidiously; it was often difficult to determine with any degree of accuracy the date of onset. Fever was present in all the cases, at times quite low, at times (usually later in the disease) high and intermittent, with or without chills. In the cases in which splenic infarctions dominated the clinical picutre the temperatures were usually high and markedly intermittent. Malaise and gastric or intestinal disturbances were frequently present; sweats occurred in nearly all the cases, usually during the latter part of the disease. In all of the cases there was progressive weakness and emaciation. The spleen was nearly always enlarged and palpable. Pains were quite constant. In some cases they were localized in

[1] Read at a meeting of the Association of American Physicians, Washington, D. C., May 3 and 4, 1910.

[2] Lenhartz, Die septischen Erkrankungen, Hoelder, Vienna, 1903; Litten, Zstchr. f. klin. Med., 1881, xl; Berl. klin. Woch., 1899, No. 28; Verhandl. d. Cong. f. Inn. Med., 1900; Osler, Quart. Jour. Med., 1909, ii, 219; Horder, Quart. Jour. Med., 1909, ii, 289; Rosenow, Jour. Infect. Dis., 1909, vi, 224; Billings, Arch. Int. Med., 1909, iv, 409; Schottmueller, Münch. med. Woch., 1909, lvii, 617.

the bones, joints or muscles; in others they seemed to be neural in origin. Mild joint swellings were not infrequently met with. At times the pains were due to the development of embolic aneurysms,[3] which are so apt to develop in the disease under discussion. The painful erythematous cutaneous nodules, the diagnostic value of which has been particularly emphasized by Osler, seem to be pathognomonic. A symptom to which no attention has been paid is the tenderness of the lower part of the sternum that was present in nearly all of our cases. It is present at times even before the anemia is sufficiently developed to account for it.

The progressive anemia of the disease and the color of the face (waxy, dirty waxy, or whitish) are noteworthy features. The leukocytes were not usually increased to any degree except when the disease was advanced and various secondary lesions occurred. Petechiæ occurred in nearly all of the cases at some time or other. At times they were abundant, and were scattered over a large part of the body. At other times they were few in number and but few crops appeared. Hematuria, microscopic or macroscopic, occurred quite commonly.

None of the cases that we succeeded in following recovered. We performed autopsies in 19 cases. In 15 of these blood cultures had been made during life; in 1 of these 15 cases the cultures were all negative (5 mere made).[4] The process was always engrafted on a previously diseased valve, and the history nearly always pointed to earlier attacks of rheumatism. The lesion was vegetative in type, not ulcerative. When the aortic valve was involved there was a marked tendency for the process to spread down and attack the wall of the ventricle, the ventricular surface of the aortic flap of the mitral valve, and the chordæ tendineæ. If the mitral valve was involved, the process spread over the wall of the left auricle and involved the chordæ tendineæ. The latter were often torn. At times the auricular lesion was much more extensive than the valvular lesion. This accounts for the fact that in many cases the cardiac auscultatory phenomena change so little during the course of the disease.

The distribution of the lesions was as follows: In 10 cases the mitral valve alone was implicated; in all of these there was auricular endocarditis. In 2 cases the aortic valve alone was involved. Both valves were affected 7 times. In 6 of these auricular endocarditis was present (in one no note is recorded as to whether there was such a lesion or not). It is evident that a lesion of the

[3] John Hopkins Hosp. Bull., 1906, xvii, 223.

[4] After this patient left Mount Sinai Hospital he was admitted to the Sydenham Hospital. Several blood cultures made there were also negative. A postmortem examination was made, and the lesions typical of subacute infective endocarditis were found. The vegetations showing masses of streptococci; no cultures were made. We are indebted to Dr. B. S. Oppenheimer for these facts.

auricle practically always occurs when the mitral valve is involved. In one case, that of a man dying of decompensation and chronic nephritis, changes were found that indicate that the lesions in subacute infective endocarditis may heal, at least to a certain extent. The characteristic lesions were found on the auricular wall, undergoing organization; the chordæ tendineæ showed the usual lesions, undergoing fibrous and calcareous changes. There were no bacteria in the blood nor in the vegetations. As a rule, the vegetations in this disease contain innumerable organisms.[5]

As stated above, we have encountered 43 cases which were characterized by the clinical course and the pathological findings just detailed, and in 36 of these cases blood cultures were made.[6] Altogether 103 examinations were undertaken.[7] In 35 cases we found the atypical cocci which we shall presently describe. In one case we found the influenza bacillus. In this case the clinical course and pathological findings were essentially the same as in the large group in which cocci were found. Even the typical, painful, erythematous nodules were present. The blood-plates in the case were remarkable in that the hemoglobin was attracted, as it were, to the colonies. For the colonies became deep red after a few days, and the rest of the plate became lighter in color.

The number of colonies found in this case was at first 53 to the cubic centimeter; later it rose to 94. In cases due to infection by the atypical cocci there was a great variation in the number of organsims present. In one case only two colonies were found in 25 cubic centimeters of blood. The largest number was 500 in a cubic centimeter. It is noteworthy that in a few cases it was found necessary to make several cultures before a positive result was obtained. And we encountered one case (mentioned above) in which five cultures made in many media were all negative. Drs. Fried and Sophian, of our laboratory staff, made a microscopic study of the blood in a number of cases,[8] and found that this gave no definite information in cases which at the time gave negative cultural results.

We were recently surprised to find that it is at times necessary to observe the plates for six days. We had usually kept them for five days. The probability is that this accounts for some of the negative results. As a rule, the organisms can be found after forty-eight hours, at times after twenty-four hours. It is advisable to use both fluid and solid media.[9] Media containing glucose at times gave

[5] The lesions in the spleen and kidneys are also quite characteristic, but will not now be discussed.

[6] Two cases are still under observation.

[7] We do not include a few cases which were studied bacteriologically after death only, because ordinary bacteria may cause a secondary or terminal infection in cases due to the atypical cocci and crowd the latter out.

[8] These results will be published later. They constitute a part of a large study of the blood or bacteria by direct microscopic examination.

[9] Libman, loc. cit., p. 216.

a better result. Serum-glucose-agar was often found to be the optimum medium.

Before proceeding to describe the cocci found in our cases, it is necessary to discuss briefly the character of pneumococci and streptococci, which they resemble. Based on the studies of Dr. Buerger,[10] we had for several years made use of certain criteria for the identification of these organisms. When Neufeld's[11] publication appeared claiming that pneumococci and Streptococcus mucosus were dissolved by bile, and that streptococci were resistant, Dr. J. Rosenthal and one of the writers (Libman) made a study to determine whether this held true according to our classification.[12] We found that all organisms which we identified as pneumococci or as Streptococcus mucosus were promptly dissolved by bile, and that those identified as streptococci were not. We included in our study a careful observation of the growth of the organisms on the surface of blood plates, the method of differentiation first suggested by Schottmueller.[13]

The earlier studies made in our laboratory by Dr. Buerger had given us the following data. Pneumococci often possess a capsule the morphology of which is diagnostic.[14] At times the capsule degenerates and resembles the one sometimes found on streptococci, or the capsule may be lost. Streptococcus mucosus also possesses a diagnostic capsule, which, however, may undergo the same changes as the pneumococcus type capsule. Ordinary streptococci often have capsules, but these are not diagnostic, as the degeneration of the capsules of pneumococci or of Streptococcus mucosus may produce the same form. The pneumococcus ferments inulin, as claimed by Hiss,[15] but not in every generation. Streptococcus mucosus always ferments inulin,[16] ordinary streptococci rarely do so. Pneumococci and Streptococcus mucosus were rarely found to throw out a precipitate in glucose-serum-agar,[17] streptococci always do.[18] The pneumococcus often grows in a typical fashion on the surface of serum-agar or glucose-serum-agar (so-called pnuemococcus ring colonies[19]). At times pneumococci or streptococci grow mucoid on the media, but otherwise differ in no way from the ordi-

[10] Jour. Exp. Med., vii.

[11] Ztschr. f. Hyg. u. Infectionsk., 1900, xxxiv, 454.

[12] Paper presented at the meeting of the Association of American Pathologists and Bacteriologists in 1908; not yet published.

[13] Münch. med. Woch., May 19, 1903, l.

[14] Buerger, Centralbl. f. Bakteriol,. Orig., xxxix, 20, 216 and 346; Jour. Infect. Dis., 1907, iv, 426; Mount Sinai Hosp. Rep., 1907, v.

[15] Jour. Exp. Med., vi, 317, and vii, 556; Centralbl. f. Bakteriol., 1902, xxxi, 302.

[16] Buerger, Centralbl. f. Bakteriol., 1906, xli, 314.

[17] In this test, 0.5 per cent. glucose is used, and an ascitic serum having a specific gravity of 1015 or more. The time of observation is two days. Dr. Buerger's positive results were obtained after two days had elapsed.

[18] Libman, Jour. Med. Research, 1901, i, 89.

[19] Buerger, Centralbl. f. Bakteriol., 1905, xxxix, 20.

nary forms. These organisms are called mucoid pneumococci and mucoid streptococci.

In the course of the studies made by Dr. Rosenthal and ourselves, we investigated 19 strains of pneumococci, 69 of streptococci, and 12 of Streptococcus mucosus. The organisms were isolated from local infections in various parts of the body. Our investigations led to the following conclusions:

1. The type capsule of the pneumococcus and that of Streptococcus mucosus, as described by Buerger, are diagnostic. All organisms presenting such capsules are dissolved by bile.

2. Solution by bile is diagnostic of the pneumococcus and Streptococcus mucosus. This latter organism is easily distinguishable by its type of capsule and certain other features.[20]

3. The absence of precipitation speaks almost absolutely against an organism being a streptococcus.

4. The fermentation of inulin is not of pathognomonic value. While it is a feature of the pneumococcus, the property may be absent, and, furthermore, streptococci occasionally give a positive result.

5. The ring colonies are diagnostic of the pneumococcus.

6. The growth on blood plates[21] gives the following results of value: (*a*) A definite large (two to three millimeters in diameter), perfectly clear zone (so-called hemolytic zone) about a colony is diagnostic of streptococci if it is found to persist in several generations. Incomplete clearing is of no diagnostic value, nor is a linear clearing. (*b*) Pneumococci always grow green[22] (once the growth was brownish green). An organism not producing a green color always proved on further study not to be a pneumococcus.

The conclusions we arrived at in the study just detailed were based on the following findings:

1. Type capsules were found on all nineteen pneumococci studied, either in the original material (pus, sputum, etc.) obtained from the patient or in cultures. All the organisms were dissolved by bile. Type capsules were found on all the strains of Streptococcus mucosus. Of 69 ordinary streptococci, 32 had capsules.

2. All of the streptococci precipitated. In one, this was slightly marked, but was intense after passage through one mouse. Of 19 pneumococci, only two precipitated; these two had been in bile (this seems to favor the development of a precipitating power) and did not precipitate in the second generation. Of twelve strains of Streptococcus mucosus, one precipitated for four generations, and one for two generations.

[20] Centralbl. f. Bacteriol., 1906, xli, 314.

[21] In this work, besides using human-blood plates, we also made use of the blood plates made of ox blood according to the method of Bernstein and Epstein, Jour. Infect. Dis., 1906, iii, 722.

[22] In our work we included, as producing green color, organisms the colonies of which either were green or were surrounded by a green zone.

3. Of nineteen pneumococci, all fermented inulin except two (in two others the result was very slight). With these two a slight result was obtained by passage through two mice. The strains of Streptococcus mucosus all fermented rapidly. Of the 69 streptococci, 2 fermented inulin, one lost the property in the second generation and also after passage through one mouse. The other lost the property after passage through two mice.

4. On blood media, all but 11 of the streptococci grew in fine gray colonies surrounded by an absolutely clear zone[23] (as described by Schottmueller for Streptococcus longus). Of the 11 exceptions, 7 gave a good white, moist growth without any clear zone,[24] 3 grew green like the pneumococcus, and one gave only a slight almost colorless dry growth with no clearing.

All the pneumococci grew in green colonies, some with partial clearing, some with no clearing. Some colonies were green by transmitted light (not by reflected light). One produced brownish-yellow-green colonies, and one produced clear zones about the colonies in a plate made with the blood of the patient from whom the organism had been isolated, but not on other blood plates. All of the strains of Streptococcus mucosus produced green colonies and were mucoid in the earlier generations.[25]

The strains of ordinary streptococci which grew white were obtained as follows: Three were from cases of otitis media and mastoiditis, one from a case of cholecystitis, one from a case of liver abscess, one was a postmortem invader.

The streptococcus which gave a slight dry growth was obtained from a skin infection. The three strains that gave green colonies were obtained as follows: One from a case of appendicitis, one from a case of otitis media, one from a specimen of sputum.

The cocci found in the cases of subacute and chronic infective endocarditis during the last few years were carefully studied along the lines we have just discussed. These organisms are all Gram-positive. They have never been found to have a capsule. They are either round, ovoid, lancet-shaped, or bacillary in form, and may be found in diplococcus form, in groups, or in chains. Every organism except one was found sooner or later to grow in chains which were often very long and convoluted. They are usually smaller than the ordinary streptococci and pneumococci, and grow more poorly, especially when first isolated. We shall not now describe their manner of growth on the ordinary laboratory media (gelatin, agar, potato, milk, etc.) because no definite diagnostic criteria are thus afforded.

[23] One of us (Libman) has pointed out that such clearing may occur in the form of concentric rings.

[24] These would correspond, at least in part, to the type called Streptococcus mitior or viridans by Schottmueller.

[25] In the paper to be published by Rosenthal and Libman, the growth of Streptococcus mucosus and of the pneumococcus will be described in more detail. A very interesting type of growth not hitherto noted was seen a number of times.

As will be seen from the accompanying table, these organisms fall into two groups, one of which ferments inulin and the other not.[26] The former comprises about one-third of the organisms. They all produce a precipitate in serum-glucose-agar and are all resistant to the action of bile.

	Pneumococcus.	Streptococcus.	Streptococcus mucosus.	Endocarditis cocci.	
				A	B
Capsule	1. Diagnostic or 2. Degenerate or 3. Not present	1. Non-diagnostic or 2. Not present	1. Diagnostic or 2. Degenerate or 3. Not present	Not present	Not present
Gram	+	+	+	+	+
Blood plates	Green; no clear zone	1. Gray; clear zone 2. Green; no clear zone 3. Moist, good white growth, or slight dry whitish growth; no clear zone.	Green; usually mucoid; no clear zone	1. Green; no clear zone. 2. Moist good white growth, or slight dry whitish growth; no clear zone	
Inulin fermentation	+ (occasionally—)	— (rarely +)	+ — (rarely +)	+	—
Precipitation.	— (rarely +)	+		+	+
Solution by bile	+	—	+	—	—

Note: Ring colonies of the pneumococcus are diagnostic.

In describing the growth of these cocci on blood plates a distinction must be drawn between the phenomena seen in the plates made with the patient's blood, and in subsequent inoculations on plates made with other blood. In the former, a definite clear zone is sometimes, although rarely, seen. It usually takes two to four days to develop. (Ordinary streptococci that produce a clear zone do it more rapidly.) In subsequent transplantations this is lost. The colonies in the original plates are usually white in color, with or without a green or an opaque zone about them. The deeper colonies are white or opaque. In subsequent cultures on blood plates, three types of growth are seen. There may be a production of green pigment, there may be a moist white growth, or a dry almost colorless very slight growth.

Before finally coming to the question of the grouping of these organisms, it is essential to draw attention to the remarkable changes which pneumococci (and to a much less extent streptococci) may

[26] A study of the growth on other carbohydrates gave no useful differential features except when raffinose was used. The organisms can probably be further subdivided by a study on raffinose media. The inulin and raffinose fermentation generally go hand in hand. Twice we found organisms that fermented raffinose and not inulin.

undergo in the body, especially in the blood current. Studies made by Drs. Buerger and Ryttenberg[27] demonstrated that pneumococci may lose their type capsules, may lose the property of fermenting inulin, and may acquire the power of precipitation.[28] One of us had previously shown that they could acquire temporarily the property of producing a clear zone about the colonies.[29]

By inoculation of such cultures into mice, the method usually employed in an attempt to bring back the feature of altered pneumococci, some organisms could be converted into typical pneumococci. However, if such organisms were first kept in media for a long period, and then inoculated into mice, the pneumococcus character could no longer be developed. As regards streptococci, we have already stated that in the work done with Dr. Rosenthal, we found two that fermented inulin, and lost the property after passage through mice. We did not find any streptococci that lost the power of precipitation, and the streptococci that produced clear zones about the colonies retained this property indefinitely.

It will now be of interest to compare the endocarditis cocci with pneumococci and streptococci. Except that they are smaller and grow more poorly (unimportant points when it comes to a question of classification), the group that does not ferment inulin resembles the type of streptococci which produce no zones of clearing in blood plates. Of course, the real relationship of these streptococci to the ordinary streptococci that produce a clear zone on blood plates has not been definitely determined. The inulin fermenting group might be considered to be a separate type, or might be regarded as being the same as the others except that they acquired the property of fermenting inulin in the body and permanently retained the property.[30] But we have no proof, as yet, that this may occur.

At first sight these organisms, especially those that do not ferment inulin, would appear to be far removed from pneumococci. But we have seen that pneumococci may lose their capsules, may lose the property of fermenting inulin, and may acquire the property of precipitation. Many of the organisms grow on blood plates like pneumococci. But we have not hitherto met with pneumococci which, when isolated from the blood, were resistant to the action of bile. If we should meet with such pneumococci, we could more easily suspect that the organisms under discussion are altered pneumococci. Possibly they represent more than one type of organism. We believe it would be best not to consider these organisms changed

[27] Jour. Infect. Dis., 1907, iv, 609.

[28] In the study made by Buerger and Ryttenberg, a study with blood plates was not systematically made. Nor were bile tests performed, the method not yet having been introduced.

[29] Libman: Johns Hopkins Hosp. Bull., 1906, xvii, 218; Trans. Path. Soc., New York, December, 1905.

[30] There was no relationship found between the type of growth on blood media and the occurrence of inulin fermentation.

pneumococci until one succeeds in having them acquire the typical pneumococcus features.

The features of the endocarditis cocci have remained constant in subinoculations carried over a long period of time (in some instances as long as eighteen months). Every organism that fermented inulin still does so. After repeated animal inoculations (mice, rabbits) no change in the important characteristics has been demonstrated. Capsules have never been developed. We have made studies of the agglutinative power of sera from patients and of sera of animals immunized by repeated injections of the cocci, but we have thus far not found any agglutination of pneumococci or streptococci.[31] Studies made by Dr. Fried of the opsonic power of patients or of immunized animals have given no definite results. Nor have the studies made up to the present time by Dr. Kaliski and Dr. A. Sophian by means of complement fixation tests.[32]

We might add here that the organisms are not, as a rule, very pathogenic for animals. In mice we have sometimes produced peritonitis by intraperitoneal inoculations. In rabbits we have succeeded in producing peritonitis, and several times have produced endocarditis. In one instance the vegetations that were produced almost blocked the aortic orifice. Endocarditis resulted from our experiments in a much smaller proportion of the inoculations than in those of Rosenow.[33] This held true even after we adopted the same methods as were used in the work of this observer.

Let us now glance at the results obtained by other observers in cases of subacute infective endocarditis, in order to ascertain what organisms they found and whether any of the organisms were the same as those which we encountered. An accurate comparison is not possible, because no two authors have made use of the same methods in studying the organisms.

Lenhartz,[34] in 16 cases, found streptococci 13 times, a staphylococcus, a pneumococcus, and a gonococcus, each once. He says that in 10 of the 13 streptococcus cases the organism was small. He calls it "Streptococcus parvus." No further characteristics are given. We believe that some of his organisms must be the same as some of ours because they are found in the blood in the same type of disease.

Osler[35] describes 10 cases, in 6 of which blood cultures were made. Three of these gave a negative result, one revealed a streptococcus and one a staphylococcus.

Horder[36] in a very extensive and thorough study of various forms of infective endocarditis, describes 13 cases, from 9 of which he isolated streptococci, and from 4, influenza bacilli. He identifies

[31] These studies were carried on in association with Dr. A. Sophian.
[32] All these investigations will be further prosecuted, as only positive results are of value.
[33] Loc. cit. [34] Loc. cit. [35] Loc. cit.
[36] Loc. cit.

these streptococci with "Streptococcus salivarius" and "Streptococcus fecalis," two of five groups into which Andrewes and he[37] had classified streptococci (the other three groups are Streptococcus pyogenes, Streptococcus anginosus, and the pneumococcus). This classification is based mainly on the differences in the power of different streptococci to ferment various carbohydrates. But some earlier work done in our laboratory[38] and some recent studies on this subject by Dr. Sophian and ourselves indicate that the classification of these authors can not be adopted. Whether Horder's organisms are the same as ours it is not possible to say, because the methods of study used by him and by us differ. Considering that the clinical course was the same as in our cases, the organisms may very well have been identical. It is of interest in connection with our results to note that he found not only organisms classed as streptococci, but also influenza bacilli in the blood of his cases.

Rosenow[39] in a contribution to this subject replete with valuable bacteriological data, to only a few of which we can now refer, states that he found pneumococci in all the cases that he studied. "When the organisms were first isolated in broth they grew in clumps and produced a diffuse turbidity slowly, and upon agar slants the colonies grew more or less tightly to the surface. The stained specimens gave diplococcus forms and chains. The chain formation as well as the property of growing in clumps in broth and of adhering tightly to the surface of blood-agar slants disappeared after cultivation for a variable period. . . . By cultivation on artificial media all the strains change gradually into typical lanceolate diplococci, often capsulated, growing as typical pneumococci in broth and on blood-agar slants. On animal inoculations this modification often occurred abruptly." The agglutination and opsonic studies of Rosenow do suggest a relationship of his organisms to pneumococci, but until the organisms are shown to have definite pneumococcus character, they cannot be surely classed as pneumococci.

Through the kindness of Dr. Rosenow we were enabled to study five of his organisms (Nos. 292, 293, 362, 409, 413). We have not yet finished a comparative study of his organisms and ours. Only one of the organisms now produces a green color on blood plates, and that is very slight. One organism (409) ferments inulin regularly; all of the organisms are insoluble in bile. Capsules can not be demonstrated by us. As regards chain production (on which we are not willing to lay any great stress in diagnosis), it could be observed in the growth of all the organisms in fluid media. Three of the organisms precipitate markedly, two slightly. The organism that precipitates actively (409) is also the inulin fermenter.

[37] Lancet, 1906, lxxxiv, 708; see also Gordon, Lancet, 1905, lxxxiii, 1400.
[38] Buerger: Jour. Exp. Med., 1907, xix, 428.
[39] Loc. cit.

Schottmueller[40] in a recent publication, describes under the name "endocarditis lenta," the clinical features of subacute infective endocarditis. He cites 5 cases, in the blood of all of which he found the organism which he calls "Streptococcus mitior," or "Streptococcus viridans," because the colonies are those of a poorly growing organism and because they produce a green pigment on blood plates.

It will be remembered that Schottmueller classed the streptococci (apart from Streptococcus mucosus) into two groups according to the manner of their growth on blood plates and in blood bouillon.[41] The one type is "Streptococcus longus," which grows in fine gray colonies and produces a marked clearing of the media; in blood bouillon it produces a carmine or Burgundy red color. The other, Streptococcus mitior or viridans, which grows slowly, develops fine gray or black-green colonies, and produces no clearing; in blood bouillon it produces a brownish-red discoloration. No data are given as to inulin fermentation, precipitation, or the reaction with bile, three of the criteria which we consider of importance. It is, of course, very likely that Schottmueller's organisms are the same as ours. He believes that the organism is specific for subacute infective endocarditis. With this conclusion we cannot agree, as we have also found influenza bacilli in the blood in one such case, and Horder described four such cases.

We have shown the difficulties that are encountered in an attempt properly to classify the organism which we have met in our cases. We have also shown that organisms which are practically the same as the group which does not ferment inulin have been found by us in local infections of various kinds. The important point to be brought out is that up to the present time whenever we have found the organisms which we have usually called endocarditis cocci par excellence (or attenuated streptococci of endocarditis) there was always present an infection of the endocardium. In no other disease have we found these organisms in the blood current. They, therefore, seem to have diagnostic import.[42]

Our studies on the bacteriology of the blood now comprise over 3000 blood cultures, made in the last twelve years. Our attention was first drawn to the fine colonies of the endocarditis cocci by Dr. M. Gershel, then an interne, in 1902. In the following year Schottmueller drew attention, in the publication already cited, to his "Streptococcus mitior." Since 1902 we have made 2750 blood cultures. Many cases of acute endocarditis were studied in which ordinary streptococci, staphylococci, pneumococci, and the

[40] Loc. cit.

[41] Schottmueller also describes differences in the manner of growth in milk and bouillon, but, as stated before, these phenomena cannot be depended upon for definite differentiation of organisms in the streptococcus-pneumococcus group.

[42] The accurate characterization of these organisms will be of value in studying the source or sources of infection.

genococcus were found. But these organisms have not thus far been encountered in any blood culture made in the type of endocarditis to which this paper is devoted. Some of these cases present such a typical symptom complex that a clinical diagnosis can be made with a fair degree of certainty. It may, however, be difficult to differentiate them from cases of chronic endocarditis with fever in which there is no superimposed bacterial infection of the valve, and the disease is not infrequently confused with such conditions as rheumatism, typhoid fever, tuberculosis, Banti's disease, or splenic anemia.[43] While the onset is usually insidious and acute phenomena do not develop for a long time (or possibly not at all), there are cases which first present themselves with a picture resembling an acute surgical condition. Under these conditions the disease has been mistaken for appendicitis (because of acute abdominal symptoms), for abscess of the spleen, or for pyelonephritis. And it has transpired that surgical measures have been adopted in such cases.

As we have already indicated we have thus far found no help in diagnosticating the disease by microscopic study of the blood, by opsonic agglutination, or complement fixation tests. The absolute diagnosis must, for the present, rest on the cultural study or the blood.

SYPHILITIC FEBRILE PYLEPHLEBITIS, WITH REMARKS ON SYPHILITIC FEVER AND ABERRANT TYPES OF HEPATIC SYPHILIS.

By Arthur R. Edwards, A.M., M.D.,

PROFESSOR OF THE PRINCIPLES AND PRACTICE OF MEDICINE IN THE NORTHWESTERN UNIVERSITY MEDICAL SCHOOL, AND PHYSICIAN TO THE MICHAEL REESE HOSPITAL, CHICAGO.

Epitome. The following report concerns a case of acute pylephlebitis with high remittent, intermittent, and prolonged pyrexia, extreme emaciation, anemia, and weakness, and rapid recovery under antisyphilitic medication. The discussion of the case covers: (*a*) A brief consideration of the classical forms of hepatic syphilis chiefly in their relation to aberrant types of hepatic syphilis; (*b*) fever in visceral lues, particularly in hepatic localizations; and (*c*) the mimicry of septicopyemic and toxemic conditions by syphilis of the liver.

Report of the Case. The patient, Albert B., came under observation September 13, 1908.

Personal History. Male, aged twenty-five years, single and a fruit pedler. Born in Tennessee and lived in Illinois the last five

[43] Schottmueller. See also Luening, Deut. med. Woch., 1900, p. 1908. In one case a clinical diagnosis of tuberculous kidney was made because of the marked renal phenomena.

years. Drinks beer very moderately, but uses no whiskey. Smokes twenty to thirty cigarettes a day, and drinks two cups of coffee with each meal. Six years ago an attack of gonorrhœa, without severe symptoms or any sequels. No venereal sores of any variety.

Family History. Father died from unknown causes. Mother died twenty years ago with pulmonary tuberculosis. Brother and sister living and healthy. No other data.

Previous Illnesses. Usual diseases of childhood, including pneumonia. Seven years ago chills and fever in Tennessee, which lasted intermittently for seven weeks and were cured with quinine.

Present Trouble. September 10, after drinking water which tasted stale, he developed malaise, severe headache, high fever, pains in the back and limbs, and went to bed. In the evening nausea was followed by three attacks of vomiting. He coughed, but no more than for the last eight months, from excessive smoking. There had been no chill, sweats, loss of weight, jaundice, urinary or circulatory symptoms, etc.

Examination. The pupils and other reflexes were normal; the nutrition was good and the skin showed no œdema, scars, or eruption. Genitalia negative. The tongue was heavily coated with a moist white fur, and the pharynx was negative. The lungs gave a normal resonance and free excursion and were free of rales. The heart examination was entirely negative, and the vessels were soft. The abdomen was retracted, but not rigid, and the edge of the liver was normal, being felt clearly on deep inspiration. The splenic margin was distinctly palpable, soft in consistency and slightly sensitive.

The patient's general appearance somewhat suggested typhoid fever, but the fever cure was irregular and a severe rigor was observed on the second day, designated by the crosses on the accompanying fever chart. A blood culture taken on the first day was negative; the leukocyte count was 6400; a Widal test was entirely negative and the urine showed no diazo reaction. The sunken, lax abdomen was repeatedly explored, but only the moderate splenic intumescence was found.

Two more blood cultures and two Widals tests were negative; the leukocyte count rose once to 11,400, but fell to 4000 on the fifth day (September 18), and the fresh and stained blood was carefully and repeatedly examined for plasmodia, but with negative results.

As indicated in the chart, the high fever elevations ceased after one week, and for six days the curve hovered just above or below normal, when suddenly, with a rigor and renewed pyrexia (September 26), the patient complained of pain over the liver, which became tender, and a massive ascites developed in the course of the day. This sudden effusion contrasted very remarkably with the abdominal retraction which had existed up to this time. With the precipitate ascites the patient vomited severely at almost hourly intervals during September 26, and the aspect of the patient was

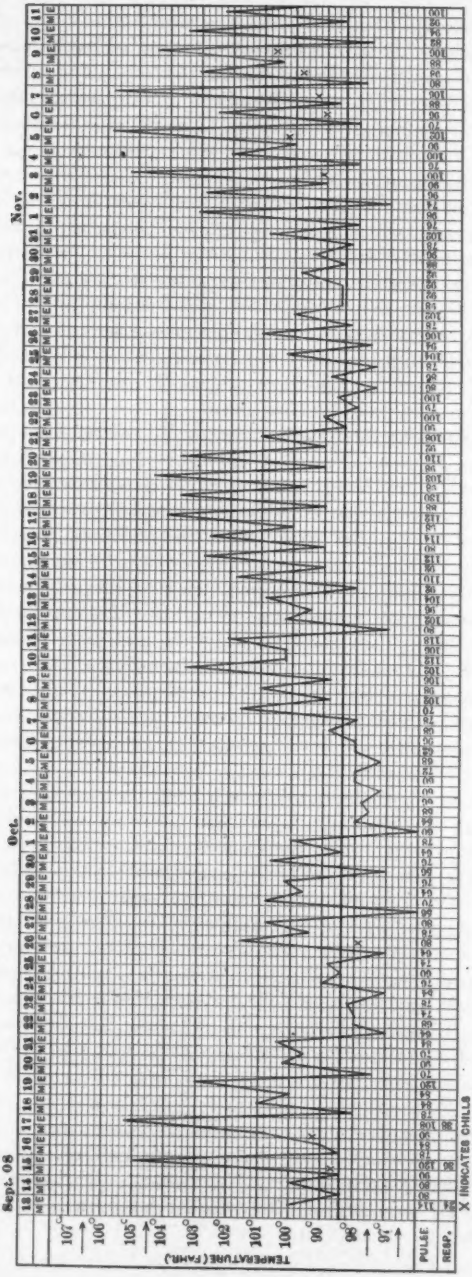
desperate, although the pulse remained slow and of fairly good quality. The possibility of a rupture of the gall-bladder, stomach, or bowel was seriously considered for the next day, and operation was advised by some who saw the patient. The leukocytes counted 6400. The urine, negative up to this time, became scanty (400 c.c.), was almost solid with albumin, and contained very many granular and hyaline casts and considerable blood.

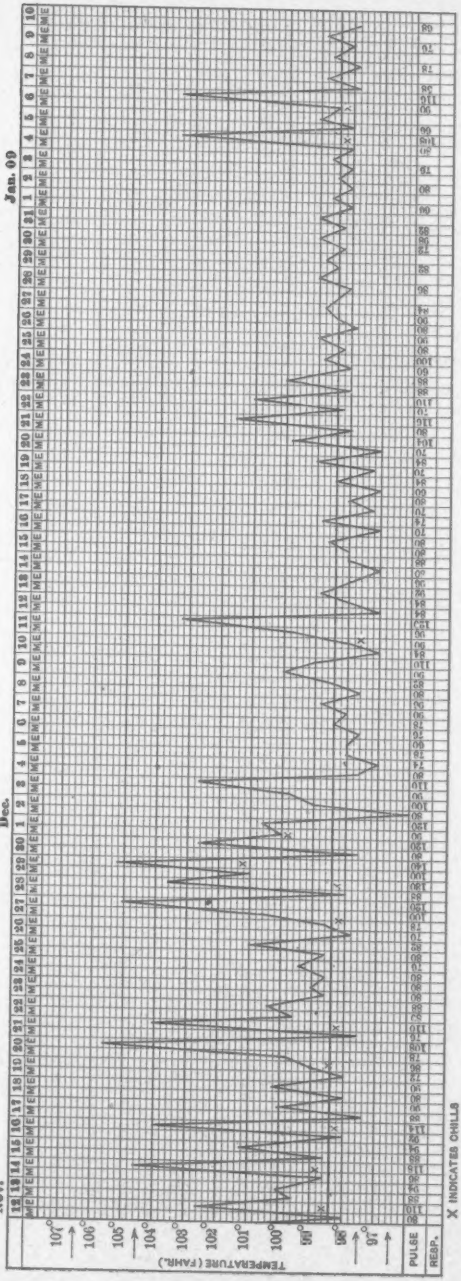
From September 26 to October 8 the fever declined, but chills and an up-and-down curve reappeared. Another Widal test and blood culture were negative. The blood showed no parasites, and an ophthalmo-tuberculin test was completely negative. The red cells counted 1,440,000, the whites 6600, and the hemoglobin was 40 to 50 per cent. The differential leukocyte count was normal, and there were no abnormalities in the stained specimen. Red and white cells, hyaline and granular casts, 3 per cent. of albumin, and a partial diazo reaction were noted in the urine.

The patient looked extremely anemic, but without the lemon tint of pernicious anemia. From October 8 to November 8 the course is best shown by the fever chart, in which a considerable remission of the fever is noted between October 20 and November 1. In the first week of November there were three febrile movements to nearly 106°, with daily variations of about 8°. What appeared to be the maximum possible distention of the abdomen increased. The blood pressure measured 135 mm. Further white counts were 8400, 6400, and 5200. Two more Widal tests and three blood cultures were negative. On November 8 a rigor lasted two hours. Vomiting and abdominal pain became more severe, and, as an after-thought of the mere possibility of lues, on November 14 the patient was put upon daily mercurial inunctions and 30 grains of potassium iodide three times daily internally.

Within three days the subjective gastric distress and abdominal pain, as well as the objective ascites, œdema, and nephritis were markedly improved. The fever, chills, and sweats were less frequent and less severe, the patient's appearance was totally different, and on November 28 the patient sat up, against orders. Point by point improvement increased, and while in December there were three, and in January two rigors and rises in temperature, the patient gained weight, color, and strength, and eighteen months later he is well and working. The Wassermann test made in January by Dr. Adolph Gehrmann was negative, but the patient then had been actively treated with antisyphilitic remedies for about two months.

The recital of such a case report is, of course, open to criticism. The disease was clearly some severe infection, but the most careful tests for its cause were negative. The brusque development of pain over the portal vein region, the sudden tenderness, and massive


Sept. 08
Oct.
Nov.
TEMPERATURE (FAHR.)
PULSE
RESP.
X INDICATES CHILLS



Fever chart of a case of syphilitic pylethrombosis.

ascites argue for acute pylephlebitis, or some sudden compression of the portal vein. Such extreme ascites certainly was not of renal origin. Inasmuch as the liver's edge had been clearly palpated, with negative findings, an involvement of the portal vein seemed a logical inference.

Naturally a septic pylephlebitis would be considered first, and this probability fortunately saved the patient from fruitless surgical intervention. After the possibility of syphilis was entertained, the immediate response to mercury and iodides was most suggestive—if a diagnosis *ex juvantibus* is ever logical. Rosenbach[1] and others have criticised diagnoses based only upon the results of therapy, but since Klemperer[2] reported the cure of hepatic enlargement attended by chills and fever, some dozen similar cases in the literature of hepatic syphilis have been described by trustworthy observers[3].

(*A*) Typical and Particularly Aberrant Types of Syphilis of the Liver. Acquired syphilis of the liver is a common malady, yet I believe that its significance is not fully appreciated either in practice or in the literature. When one reviews the great urban and rural prevalence of syphilis, and considers that we possess absolute specifics against this infection, one is surprised that he does not oftener consider the possibility of lues in doubtful cases. In the seventeenth century hepatic syphilis was described by Botalli, Petronius, and Mercurialis, and later more fully by Bonet, Portal, and Ricard, yet Dittrich, in 1849, first fully developed the topic.

Acquired syphilis of the liver exists in two main classical forms, the syphilitic cirrhosis and hepatic gummas. It is not my purpose to describe these well-known types, except so far as they may mimic other diseases, as hepatic cancer and abscess, gastric disease, gallstones, typhoid fever, sepsis, cirrhosis of the liver, and pylephlebitis.

1. *Hepatic Gummas Resembling Cancer of the Liver.* This confusion is one of the oldest, and Oppolzer and Bochdalek mistook hepatic syphilis for cancer.

(*a*) Age is no criterion, although it is commonly asserted that syphilis is prone to devolop in those under forty years of age, whereas cancer prevails largely after that year. In personal experience, syphilis is as common after as before the fortieth year, and it is not uncommon for gummas to form in the liver two to four decades after the initial chancre.

(*b*) It is maintained that in syphilis the tumors are generally smaller than in cancer, but gummas may so distend the liver that the organ occupies most of the abdominal cavity. In one very

[1] Therapie der Gegenwart, 1903–1904.

[2] Ibid., 1903.

[3] J. Mannaberg, Wien. klin. Woch., 1902, Nos. 13 and 14; L. Lippmann, Therapie der Gegenwart, 1904; Reye, Therapie der Gegenwart, 1905; J. Israel, Deut. med. Woch., 1904, No. 14; E. G Janeway, Amer. Jour. Med. Sci., September, 1898, p. 251.

emaciated patient an epigastric swelling, larger than the patient's head, subsided under mercury and iodides, which therapy was suggested by the Argyll Robertson pupils. These luetic tumors generally develop more slowly than cancer, at least after they are once detected; they remain more stationary, and compression symptoms, as crowding of the enlarged liver upon the lungs and other structures, are more suggestive of cancer than of syphilis.

(*c*) *The edge of the liver* is rather smooth, or its anterior surface, near the suspensory ligament and near its palpable edge, is lobulated by deep, radiating furrows resulting from cicatrization and organization of the gummas.

(*d*) *Ascites and Icterus.* Fournier and others have remarked that the absence of ascites is a rather safe criterion for syphilis as against, *e. g.*, cancer, and this dictum doubtless holds for most cases, but cancer will be considered first in the minority of cases in which ascites is associated with icterus and emaciation, which latter, in extreme instances, may amount to cachexia. The uncommon ascites results from syphilitic cirrhosis, from amyloid degeneration, or, as a terminal event, from cardiac weakness. Syphilitic ascites is frequently combined with albuminuria.

(*e*) *Splenic enlargement* is exceptional and follows the compression of the portal vein by a gumma or follows amyloid degeneration of the spleen or gummas in its substance.

(*f*) *Pain.* Pain on movement, pain in the right shoulder, and tenderness over the liver almost invariably indicate perihepatitis, and therefore suggest syphilis. If the perihepatitis is recent, a friction rub may be heard over the liver; if it is old, adhesions to the colon or stomach and abolition of the respiratory excursion of the liver may result. Pain is important and conspicuous both in cancer and syphilis. In two cases in which large masses disappeared under specific treatment, paroxysmal pain was probably due to coincident, though poorly developed tabes. These observations are interesting in view of the fact that there is said to be a degree of antagonism between syphilis of the liver and the parasyphilitic nervous diseases, as tabes. Search for other evidences of syphilis and a history of venereal infection are important, but these considerations are purposely subordinated, as we must frequently diagnosticate or suspect syphilis in their absence.

(*g*) *Emaciation and Cachexia.* In hepatic syphilis, emaciation may attain such an extreme degree of development that carcinomatous cachexia is diagnosticated without much hesitation. Clearly hepatic symptoms, such as tumors, ascites, icterus, and enlarged liver, combined with anemia, emaciation, and cachexia, which are probably due to metabolic disturbances, make differentiation impossible, except by the help of successful therapy by mercury and iodides. In a patient of Dr. William E. Morgan, there was an enormous ascites and extreme emaciation; an operation disclosed a

gummatous and lobulated liver, though previously carcinoma seemed almost certain. After the incision, specific remedies produced immediate and permanent recovery.

In just this group of cases gastro-intestinal symptoms may seem to indicate a primary cancer in the stomach or bowels. In one instance there were tenderness and pain in the pyloric region, vomiting, occult blood in the vomitus and feces, absence of free hydrochloric acid in the test meal, and cachexia; the presence of nodes in both lobes of the liver strengthened the probability of gastric cancer with secondary metastases in the liver. The history of venereal infection suggested the bare possibility of syphilis, and sublimate hypodermics and iodides wrought a complete cure. Quincke[4] describes an instance of dilatation of the stomach, due to gummas in the mesentery, which responded, rapidly and permanently, to iodides. Inasmuch as gastro-intestinal symptoms suggest a primary neoplasm in the alimentary tract, it is profitable to note Marcuse's observation that two-thirds of the patients with hepatic gummas exhibit early digestive disturbance.

2. *Syphilitic Cirrhosis Resembling Alcoholic Cirrhosis.* There is no absolute criterion for differentiation between syphilitic and portal cirrhosis. In syphilitic induration the size of the liver is more commonly increased than decreased, even though the process is destructive rather than hyperplastic. The new-formed connective tissue follows the portal vein and its ramifications into the liver substance, the lobules of which are more invaded than in alcoholic cirrhosis. An obliterative endarteritis may augment the damage to the liver cells, which degenerate and atrophy, particularly toward the anterior border of the liver. The surface of the liver is uneven with furrows or nodules and its edge is somewhat sharper than in the ordinary form of cirrhosis. Perihepatitis is common and results in various adhesions. Icterus occurs in one-third of the cases and the attendant enlargement of the spleen develops from stasis, toxemia, or amyloid degeneration. Ascites is less common than in the alcoholic variety, develops later, and is prone to recur after tapping. A longer clinical course and fairly good nutrition are said to be characteristic, but there are many exceptions. Two greatly reduced subjects, apparently suffering from advanced cirrhosis, recovered completely under iodides.

3. *Gummatous Hepatitis Resembling Gallstones.* Riedel,[5] particularly, has drawn attention to an important group of cases, which simulate gallstones, cholecystitis, and, if icterus is present, even resemble calculous obstruction of the common duct. Coincident temperature may further obscure the differentiation. In this type the gall-bladder is never involved alone, but there is always coin-

[4] Deut. Archiv f. klin. Med., 1903, lxxvii.
[5] Mitteilung aus dem Grenzgebiet. d. Med. u. Chir., 1904, xiv, 1.

cident syphilis of the liver. Riedel reported several such cases, and Trinkler[6] found 13 reported operations in which liver gummas were found instead of some other suspected surgical disease.

(*B*) FEVER IN VISCERAL LUES, PARTICULARLY IN HEPATIC LOCALIZATIONS. The topic of fever in syphilis dates from Werlhof (1732), J. Frank (1821), and Yvarres (1854). Güntz was the first to record syphilitic fever taken by the thermometer, and Bäumler[7] wrote upon the subject.

Fever in hepatic syphilis was described by Frerichs and Gerhardt, and within recent years Stauder, Mannaberg,[8] Klemperer, Janeway, Gerhardt[9] and others have written convincing articles which have not attracted the attention they merit. It is beside our subject to discuss the fever of initial or secondary syphilis. Under the next topic luetic pyrexia will be again considered.

(*C*) THE MIMICRY OF TOXEMIC AND SEPTICOPYEMIC CONDITIONS BY SYPHILIS OF THE LIVER. 1. *Hepatic Gummas Resembling Abscess of the Liver.* Luetic liver symptoms, as pain, tenderness, and enlargement, together with fever, chills, and other septicemic manifestations, may closely resemble liver abscess. In a patient of Dr. Lesage there was an exquisitely tender point in the epigastrium, with a leukocytosis of 21,000, chills, fever reaching 102° or 103° over a period of nine months, and a loss of 40 pounds in weight. Under mercurial injections and iodide of potash internally, every symptom subsided in a week, never to recur. Trinkler, Lenhoff, and Ebstein noted a pseudofluctuation which heightened the resemblance of liver abscess. In most of the writer's cases, the leukocyte count did not run high, but in three instances it ran 21,000, 17,300, and 15,200. The fever may be most deceptive,[10] and in hepatic differentiation fever argues scarcely more for abscess than for syphilis.

2. *Gummas of the Liver Simulating Tuberculosis, Typhoid Fever, Septicopyemia, Malaria, etc.* In the absence of any localizing visceral, *e. g.*, hepatic symptoms, fever alone may prove most deceptive. In one patient, seen with Dr. Black, the fever ran many weeks; blood cultures were negative, the Widal reaction was negative, and until the autopsy disclosed widely diffused gummas in the liver, typhoid fever, sepsis, and meningitis were considered, but without any definite conclusion being reached. E. G. Janeway[11] reported cases in which the fever simulated pulmonary tuberculosis. Bäumler described cases suggesting tuberculosis and typhoid fever, and Mannaberg described an intermittent fever mistaken for malaria and septicemia, until antisyphilitic therapy dissipated all doubt.

[6] Mitteilung aus dem Grensgebiet. d. Med. u. Chur., 1904, x, 726.
[7] Deut. Archiv f. klin. Med., 1872, ix, 397.
[8] Ztsch. f. klin. Med., 1907, lxii, 253.
[9] Berl. klin. Woch., 1900, p. 1046.
[10] Deut. Archiv f. klin. Med., 1908, xcii, 236.
[11] AMER. JOUR. MED. SCI., September, 1898, p. 251.

3. *The Prolonged Pyrexia of Pylephlebitis.* The unusually long course of the reported and almost unique case is depicted in the appended chart, and also the pyemia-like chills and steep curves.

In an analysis of the causes of pylephlebitis, Lissauer[12] found enlarged portal lymph glands in 46 per cent., splenic abscess in 28 per cent., pancreatic cancer in 24 per cent., primary hepatic cancer in 11 per cent., appendicitis in 5 per cent., and syphilis of the liver, primary gall-bladder cancer, and secondary hepatic cancer, each in 4 per cent., etc.

CONCLUSIONS. As suggested in the introductory epitome:

1. What is seemingly a suppurative pylephlebitis, with intensely septicopyemic symptoms, may be syphilitic pylephlebitis with spirochete sepsis.

2. There is always the possibility that various vague or obvious portal-vein, gall-bladder, and liver symptoms may be syphilitic—a possibility of vast therapeutic significance.

3. A diagnosis of syphilis made *ex juvantibus* may be open to criticism; while not of absolute certainty, such means of diagnosis of syphilis exceed the degree of probability.

4. Hepatic gummas may precisely simulate hepatic cancer, even to the extent of the characteristic cachexia, and perhaps, also, of the significant gastric findings.

5. Certain cases of seeming atrophic cirrhosis may respond to antiluetic remedies.

6. Cholelithiasis, cholecystitis, and hepatic abscess may be simulated by syphilis of the liver.

7. Tertiary invasion of the liver or portal vein may run its course without localizing signs, and masquerade as typhoid fever, septicemia, tuberculosis, or malaria. When the Widal reaction, blood cultures, tuberculin test, and examination for plasmodia are negative, it is not fantastic to suggest the employment of mercury and iodides.

STRETCHING OF THE CARDIA IN THE TREATMENT OF CARDIOSPASM AND IDIOPATHIC DILATATION OF THE ŒSOPHAGUS.[1]

BY MAX EINHORN, M.D.,

PROFESSOR OF INTERNAL MEDICINE IN THE NEW YORK POST-GRADUATE MEDICAL SCHOOL, NEW YORK.

LATELY[2] I reported a case of idiopathic dilatation of the œsophagus, in which, by stretching the cardia with a metallic cardio-

[12] Virchow's Archiv, cxcii, 278.

[1] Read at a meeting of the American Gastro-enterological Association, St. Louis, Mo., June 6, 1910.

[2] Report of a Case of Idiopathic Dilatation of the Œsophagus, New York Medical Journal, May 29, 1909.

dilator, constructed by myself, a cure was effected. In that paper the literature was referred to. Now I would like to report on this method of treatment in two cases of cardiospasm and in three new cases of œsophageal dilatation. If we reflect that until recently idiopathic dilatation of the œsophagus was considered by most clinicians as an incurable affection, the present paper on the subject may not be deemed superfluous.

Before entering into a description of the cases I may be permitted to add a picture of the cardiodilator as I have slightly modified it since the appearance of my first publication. The instrument (Fig. 1) is provided with a scale for the dilatable part and is made in two sizes, one dilating up to 8 cm., the other up to 10 cm. in circumference.

As to the relations between cardiospasm and idiopathic dilatation of the œsophagus, the following remarks apply: Cardiospasm is a relatively frequent affection, which occurs much oftener in the female sex, and is generally based upon a nervous foundation. The symptoms occur during or immediately after the small meal (for these patients take only a little and that cautiously and slowly). There is a feeling of pressure in the chest, frequently turning into pain. Vomiting usually does not occur. Improvement often takes place, lasting for a long period, only to yield to a return of the trouble. While the cardiospasm lasts the swallowing sound is often delayed, sometimes broken, rarely entirely absent. Even small quantities of food may cause these troubles, since they pass the œsophagus only with discomfort; therefore, we usually do not find dila-

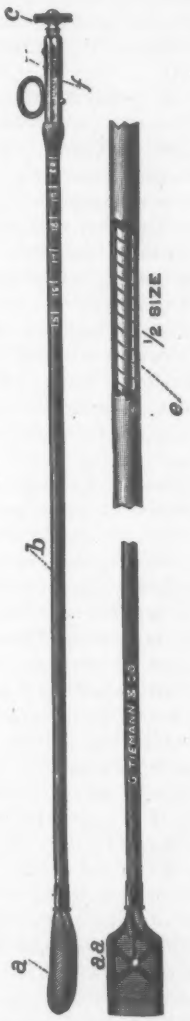


FIG. 1.—The cardiodilator. *a*, expanding end; *b*, flexible shaft; *c*, pilot wheel; *d*, handle and casing for actuating mechanism; *e*, flexible spiral shaft enclosing transmission wire; *f*, scale; *aa*, expanding end when giving maximum dilatation.

tation of the œsophagus. When, however, the cardiospasm becomes continuous, and has lasted many months or even years without interruption, a marked dilatation of the œsophagus will slowly develop.

If the latter is present, no matter whether due to cardiospasm or other causes, the clinical picture is different and accompanied by grave symptoms. The patient can apparently take a little more food without discomfort, but this is soon followed—as the œsophagus becomes distended with food—by severe symptoms. The sensation of pressure may be intensified to a feeling of choking. The patient tries to help himself by further drinking of water and compression of the thorax. If he does not succeed in forcing the food from the œsophagus into the stomach, the contents will be ejected through the pharynx and mouth. The œsophagus is rarely entirely empty, and generally contains remnants which putrefy and exert an irritation upon the walls of the œsophagus.

There now arise disagreeable sensations even between meals, and occasionally food will ooze into the larynx in the recumbent position (during sleep), which causes the patient to wake up with paroxysmal coughing. The suffering is continuous. If the patient feels better at times, he is still never entirely free from trouble; the latter is only a little less acute. The patient can never eat a meal with pleasure, but must always interrupt it, in order to help himself through the manœuvres detailed above. Objectively the swallowing sound in these cases is always absent, and an *x*-ray picture readily shows the greatly dilated œsophagus.

Before I enter upon a description of the new cases, I would like to remark that Miss Kate G., whose case was described in my paper cited above, has remained entirely well since the stretching of the cardia, and has gained about 50 pounds in weight. The cases that I have since observed are as follows:

Case I.—April 13, 1910. *Cardiospasm.*

Mrs. I. S., aged twenty-four years, has three children, the eldest three years old, the youngest seventeen weeks. Three weeks after the birth of the last child, or about three months ago, she suddenly experienced a cramp in the epigastrium. Since that time she complains of difficulty while eating. When she eats, the food seems to stick in her throat and she has the sensation of choking. By pressing she was able to get the food into the stomach. When this was not successful, she put her finger in her throat and vomited. The swallowing sound is present after eighteen seconds in an interrupted manner. Bougie No. 43 French passes the cardia. The cardia was at first stretched to 6 cm., later to 7 cm.[3] Immediately after the first stretching the patient could eat better and the swallowing

[3] Before inserting the cardiodilator it is advisable to test the screw arrangement as to its working perfectly.

sound was present at once. The patient gained in weight and felt very well.

CASE II.—March 30, 1909. *Cardiospasm and Moderate Dilatation of the Œsophagus.*

Mrs. Pauline B., aged thirty-four years, had rheumatism when eleven years old; otherwise she has been quite well. Three years ago she began to be troubled with belching of gas and difficulty in swallowing her food. It would go down as far as the cardiac orifice; then she would have a cramp and would have to straighten up and wait until she would be relieved. Sometimes it only took a few seconds, sometimes longer; again she would have to walk about in order to overcome the sensation. During the night at times a sudden cramp in the same spot would awaken her. This would last from fifteen to twenty minutes. By rubbing and drinking hot water she would feel relieved. When lying on her left side and back the cramps occur more frequently than when she lies on her right side. There is constipation, but no headaches. Solid food agrees with her much better than liquids. Acids and sour fruits are very bad.

The examination revealed atony of the stomach, right movable kidney, and slight hyperchlorhydria. These conditions not explaining her real trouble, she was examined with regard to her œsophagus. The swallowing sound was found to be absent. A whalebone bougie (54 French) encountered some resistance at sixteen inches from the teeth (cardia), but entered the stomach. The œsophagus also contained some fluid mixed with food remnants, although the quantity was small (about two ounces).

The diagnosis of cardiospasm with very moderate dilatation of the œsophagus was made, and patient treated by stretching with the cardiodilator. At the first stretching dilatation of the cardia to 7 cm. was accomplished, then up to 8 cm. for one minute. This treatment was carried out during the months of April and May, 1909. The patient was relieved from her attacks and could eat ordinary food and liquids. She remained well for about seven months, when the attacks reappeared, although in a mild degree. Her cardia was again stretched for one minute to 8 cm. on January 10, 1910, whereupon complete relief was experienced.

CASE III.—April 6, 1910. *Cardiospasm with Moderate Dilatation of the Œsophagus.*

Sister G., aged thirty years, had pleuropneumonia of both lungs about four years ago, which lasted six weeks. Last September (seven months ago), while carrying a box of candles and walking down stairs, she missed a step, and in order to save the candles she fell, and struck her left side in the region of the lower ribs. She had no pain after the fall. The following morning, on an empty stomach, she vomited coffee-ground material and had a stitch in her side (left hypochondrium). Since then she has vomited every day

(for the last seven months); once three times in the day. She always vomited small quantities of liquid. She has a pressure in the chest, as if every mouthful of food would stick there. She never experienced a sensation as though the food would suddenly slip into the stomach. Her only relief is the vomiting, which occurs practically after each meal. The vomited matter is very sour, sometimes frothy and mucous. The bowels are constipated, the appetite is poor, and the patient has lost 38 pounds since her trouble began. The swallowing sound is absent. A bougie 43 passes into the stomach. The duodenal bucket reached the duodenum and there was no blood stain present.

FIG. 2.—Radiogram of the œsophagus of Sister G., taken April 12, 1910, after ingestion of bismuth and cream. The œsophagus shows moderate dilatation.

On April 9, 1910, the cardia was dilated up to 6 cm. for one minute. She felt pain, so that it was thought best not to dilate further. Somewhat later she drank a mouthful of water, and a swallowing sound was present immediately. On April 11 the

cardia was stretched for one minute to 8 cm. The patient took her meals nicely and did not vomit.

An *x*-ray picture was taken on April 12. It shows a moderate dilatation of the œsophagus (Figs. 2 and 3).[4] Twice weekly the cardia was stretched for one minute to 8 cm.

FIG. 3.—The same as Fig. 2 a few minutes later, when the patient (Sister G.) had partaken of a glassful of milk. Most of the bismuth has passed into the stomach; there is, however, still an irregular line of bismuth visible along the œsophagus.

On April 30 patient felt very well and had gained 17 pounds in weight in the three weeks after the treatment was instituted. She ate ordinary foods without inconvenience and there was no trace of vomiting. The swallowing sound was present after six to seven seconds in a normal manner.

CASE IV.—December 8, 1909. *Idiopathic Dilatation of the Œsophagus.*

Mrs. E., aged fifty-one years, has had eight children, five being alive. She was formerly always well. After the death of her son, in

[4] I am indebted to Dr. L. G. Cole for the radiograms.

1904, which affected her a great deal, the began to feel sick. She weighed at that time 237 pounds; and lost 110 pounds up to October 1. At first in working, a little food would rise without any pain; she never had trouble in swallowing. From the very start the food rises just as it was swallowed. She was always hungry, but never ate much, because it always rose; pressing did not help it any; the attempt to bring the food into the stomach with the aid of drinking coffee did not succeed. Oil also was of no avail. Nothing ever ran out on lying down. Every morning immediately after rising, a cupful of fluid, never solid food, came up; never blood. Massage and deep breathing did no good. Even up to within a short period there were days when the patient could bring down potatoes or vegetables or cake in company. Sometimes, however, food would come up again. When excited it would be worse. The patient was never sick, nor was there any trouble in her family. During the first three months of her pregnancy she had a spasmodic cough without expectoration. She never had dryness in the throat or diminution of the quantity of urine. When she came to the German Hospital she could not even partake of fluid food, and was suffering in a high degree. She was admitted to the surgical division and Dr. Stetten kindly asked me to see the patient.

Examination showed a high degree of emaciation, the heart and lungs apparently normal, and nothing pathological in the abdominal organs. The swallowing sound was absent. Examination with the sound showed the following: The œsophagus contained about 300 c.c. of fluid, mixed with mucus and food of neutral reaction; in pushing the tube farther down it entered the stomach and a fluid containing a large amount of HCl was obtained. In using a whalebone bougie (43 French) I found a slight resistance at 16½ inches (cardiac orifice), which, however, was overcome, the sound passing into the stomach. The diagnosis of idiopathic dilatation of the œsophagus was made. (In looking over my records I found that the patient had consulted me once about two years ago, and that at that time the diagnosis idiopathic dilatation of the œsophagus was made.)

At first the cardia was stretched up to 8 cm. twice weekly for one minute; later it was done once weekly. Immediately after the first stretching the patient could take food without feeling any discomfort. She gained rapidly in weight, so that three months later she had gained 40 pounds. The swallowing sound was then present.

CASE V.—May 10, 1902. *Idiopathic Dilatation of the Œsophagus.* Clifford V. B., aged twenty-six years, suffered for two years from pains in his chest and eructation of food. In 1901 he had typhoid fever, which reduced him still more. In 1902 he weighed 125 pounds, felt weak, complained of cramps in his chest, and vomited frequently.

Examination of the chest and abdomen revealed nothing abnormal. In the fasting condition the swallowing sound was not present. A sound introduced into the œsophagus brought up 140 c.c. of fluid with food remnants, of acid reaction, no HCl. The tube was pushed farther into the stomach and a few cubic centimeters of fluid mixed with HCl was evacuated. If the patient drank a glassful of water, the entire quantity mixed with a little mucus could be obtained from the œsophagus after five minutes.

The diagnosis of idiopathic dilatation of the œsophagus was made and the usual treatment followed. The patient gained in weight, and felt better, but the difficulty of eating remained.

In 1904 I constructed a special instrument for this patient in order to dilate the cardia. The stiff part, however, was too long, and its introduction did not succeed. Thus, the patient lived on, never able to enjoy a meal like others, always having to interrupt his meal in order to force the food down by certain manipulations.

On April 14, 1909, the patient was again examined. A condition similar to that described was found. The swallowing sound was absent. The tube introduced into the œsophagus (15 inches) brought up 200 to 300 c.c. of fluid containing food (reaction neutral or faintly acid; HCl = 0; ferments absent). On pushing the sound farther down (21 inches), stomach contents, with presence of free HCl, appeared. At that time the patient weighed 137 pounds stripped. On April 20 I began to dilate the cardia, at first up to 7 cm., later to 8 cm., then 9 cm., and finally 10 cm. Immediately after the first or second week of this treatment the patient could eat more easily and he soon was able to eat larger meals in the company of his friends without having to interrupt his meals. He steadily gained in weight, and in December weighed 177½ pounds, stripped. He had gained in seven months of treatment 40½ pounds in weight. The swallowing sound could then frequently be heard six to seven seconds after the drinking of water. Occasionally the œsophagus was entirely empty. After drinking 200 c.c. of water I could obtain after one minute only 50 c.c. from the œsophagus.

Particularly instructive are the *x*-ray pictures of the œsophagus. Fig. 4 is a radiogram of the œsophagus on January 29, 1907, before the treatment of stretching began, and Fig. 5 is that of January 14, 1910, about eight months after this treatment was instituted. It is evident that the transverse diameter of the œsophagus has become markedly smaller. Fig. 6 shows the œsophagus in the lateral position after partaking of thick bismuth mush. The peristalsis of the œsophagus can be seen. The patient then drank a glassful of milk, and another picture was taken in the same position immediately afterward. The œsophagus had emptied itself completely (Fig. 7).

Epicrisis. Adding the case described before, I have now six cases of cardiospasm and idiopathic dilatation of the œsophagus

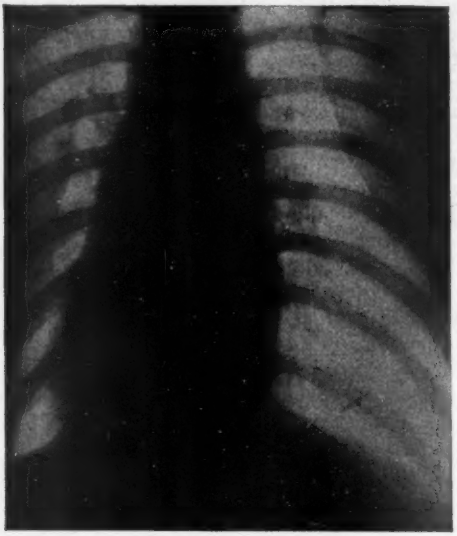


FIG. 4.—Radiogram of C. V. B.'s œsophagus after taking bismuth and cream. January 29, 1907.

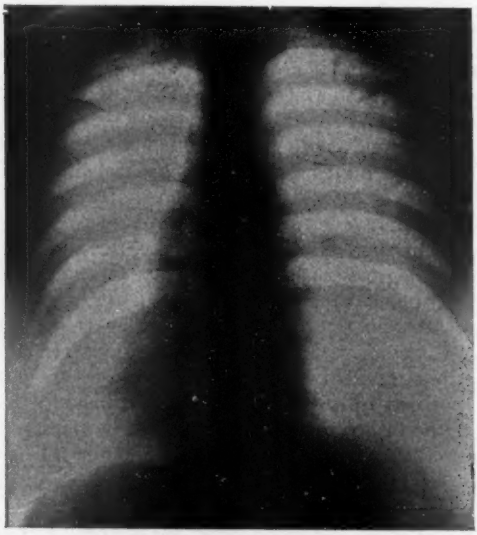


FIG. 5.—Radiogram of C. V. B.'s œsophagus after taking bismuth and cream. January 14, 1910.

FIG. 6.—Radiogram of C. V. B.'s œsophagus after bismuth in the lateral position. January 14, 1910.

FIG. 7.—The same as Fig. 6, after drinking a glassful of milk a few minutes after the bismuth, lateral position. January 14, 1910.

(two cardiospasm, four idiopathic dilatation of the œsophagus), which by means of forced stretching of the cardia were either entirely cured or so much improved that they may be considered almost cured. For even if a small residue is found in the œsophagus in some of these patients, still they do not experience any discomfort at meals and can partake of them in the same manner as normal persons. Case V (C. V. B.) deserves special mention, because the *x*-ray photographs before and after treatment by means of stretching show plainly the variation in size of the œsophagus, which is only one-third as large as before treatment. The success of treatment by means of stretching is very marked, and may now be regarded as the accepted mode of treatment in this disorder. These favorable results obtained by means of forced stretching of the cardia in cases of cardiospasm and diffuse dilatation of œsophagus have induced me to try a similar method of treatment in pylorospasm. I have, indeed, had apparently good results in some cases from stretching the pylorus.

A PRELIMINARY NOTE ON MEDICAL USES OF RECTAL INFUSIONS.

By Henry Sewall, Ph.D., M.D.,
PROFESSOR OF PHYSIOLOGICAL MEDICINE IN THE DENVER AND GROSS MEDICAL COLLEGE, DENVER.

The use of the lower bowel as a channel for the introduction of fluids into the body dates back to early days in medicine, but since rational application of the method was devised by J. B. Murphy,[1] it has become one of the salient procedures in postoperative surgical therapeutics. It is not surprising that the practice of proctoclysis, which has become well nigh routine in the surgical clinic, should promise to assume a definite place in the wider field of medical treatment. But the procedure must degenerate into mechanical manipulation unless the indications for its service and the limitations of its usefulness are established by critical observation. An intelligent application of the method presupposes a definite notion of its mode of action and of the objects to be accomplished. From a theoretical point of view the following summary may be made of the distinctive vital relations of proctoclysis: (1) Rectal infusions would seem less disturbing to the economy than fluid offered to the body through any other channel. Fluid introduced into the lower bowel by the "Murphy method" produces a minimum of peristaltic reaction, and its absorption is probably regulated by the vital demands of the organism. (2) Warm rectal infusions have a diuretic

[1] Perforative Peritonitis, Surgery, Gynecology, and Obstetrics, June, 1908.

effect, which is frequently immediate and intense. In certain cases the amount of fluid expelled from the body by diuresis greatly exceeds that introduced by proctocylsis. (3) Soluble toxins proceeding from foci of infection impair the vitality and reduce the resistance power of the healthy tissues through which they circulate. The surgeon has a lively sense of the gravity of this incident as influencing tissue repair and general reaction after operation. The condition is no less important in medical cases of disease. Dilution of the circulating toxins with imbibed fluid and their removal by diuresis would seem not only to protect the kidneys by lessening the concentration of the poisons excreted, but tend to safeguard the sound tissues from intoxication. (4) Clinical observation indicates that microbic metastasis is prone to seek out regions where tissues have already been injured by circulating toxins. Thus, in gonorrhœal infection there is reason to believe that joint pains due to transported toxins are apt to precede the local establishment of the cocci. Proctoclysis appears to ameliorate such pains and, inferentially, limit extension of the infection. (5) The addition of foreign fluid to the circulation is an important aid to the maintenance of a normal arterial blood pressure, possibly not so much by reason of the volume of fluid added as by reducing intoxication by the mechanism of circulation. (6) The exhibition of many medicines by way of rectal infusions commends itself as free from obvious disadvantages inherent to the oral channel.

For many years I have occasionally applied one or another method of proctoclysis in the medical clinic, but only during the last year has the method been somewhat systematically pursued according to the principles of Murphy's technique. In my observations the attempt has purposely been made to achieve results with the aid of such simple apparatus as is available in any bedroom. It is probable that the results would have been more satisfactory with a more adequate equipment. The fluid infused was tap water or normal saline solution, as the condition of the patient seemed to demand. One or two pints of fluid, rather hot to the touch, would be placed in the bag of a "fountain syringe" and introduced into the rectum through the hard rubber tip commonly used for enemas. The bag was usually suspended so that the surface of fluid within it should be elevated six to eight inches above the anus. The bag is raised or lowered according to the rate of outflow or complaints of the patient. Nearly two pints of fluid should flow out in the course of an hour. With this simple apparatus considerable difficulty is experienced from the cooling of the fluid in the outflow tube. This is partly remedied by keeping the tube under the bedclothes, or occasionally withdrawing the tip and allowing the cooled fluid to run off. The temperature in the reservoir may be maintained by the addition now and then of small quantities of very hot water or saline solution. As is well known, the essence of Murphy's method lies in

the free communication between the rectum and reservoir with the hydrostatic balance so slightly in favor of the latter that, while fluid surely gravitates toward the bowel, a slight excess of pressure within the rectum is relieved by a reversal of the current into the reservoir. The head of pressure in the reservoir should be so low that the distention occasioned by the flow should neither provoke contraction of the gut nor cause perceptible reaction of the anal sphincter. The special form of outflow tip, having a sharp bend in its course, which Murphy devised for patients treated in the Fowler's position does not seem necessary for medical patients that lie supine.

In my experience proctoclysis pursued in this manner is not always so indifferent to the comfort of patients as surgeons seem to find. Both the mechanical relations of the Fowler's position and the psychic distraction of a surgical injury probably combine to facilitate the application of the method in operative cases. With medical subjects it is often difficult to adjust the apparatus so as to avoid discomfort. The nurse is prone to raise the reservoir of fluid too high.

In practice it has been my habit to judge the utility of the method largely by the reaction of the kidneys. Abundant diuresis has been accepted as a sign of the efficiency of the procedure or vice versa. Instead of continuous proctoclysis, as commonly employed by the surgeon, I have usually sought the absorption of a quart of fluid twice daily, in forenoon and afternoon.

The cases on which this article is based may be divided into two groups: (1) Those in which it is desired to raise arterial blood pressure or, by stimulating diuresis, to deplete an effusion. (2) Those in which it is endeavored to reduce toxemia.

In the first group two cases gave such striking results as to form a basis for further observation. One was that of a man of middle age, probably an old alcoholic, prostrated with an asthenia of uncertain origin. Proctoclysis to the extent of two quarts of saline solution daily seemed an important aid to his gradual recovery. The other was that of a man of about sixty years, with pleural effusion, general dropsy, and orthopnœa of myocardial origin. Hot water given by the "drop method" resulted in a progressive diuresis, which in a few days removed the dropsy and led to recovery of cardiac compensation. Other attempts to reduce dropsical effusions by proctoclysis have not been so successful, but the conclusion from a considerable experience favors the exhibition of hot water by the bowel in an effort to reduce dropsical accumulations which do not yield to other remedies. Of course, a comparison of the volume of fluid absorbed with that excreted quickly determines the availability of the method.

But it is the second class of cases, those suffering from constitutional toxemia, which offer the most pertinent field for proctoclysis. Already the method has been advocated, more or less modified, in the

treatment of typhoid fever and of scarlet fever. Durbin[2] has long used colonic saline irrigations with favorable results in the treatment of typhoid fever, and Riesman[3] has recently reported satisfactory results obtained by the Murphy method. My own experience with proctoclysis in this disease has also been favorable, but is based on only a half-dozen cases. In a similar number of cases of lobar pneumonia, one of which was in an alcoholic with extensive lung involvement, high temperature, and wild delirium, I have been much impressed with the favorable effects when proctoclysis with hot tap water was made a therapeutic measure. The procedure was usually followed in a few hours by an abatement of the signs of toxemia, and especially by improvement in the mental condition.

Indeed, no class of cases has been more obviously ameliorated than that suffering from delirium. I have been struck with the rapid clearing up of the mental condition in obstinate cases of delirium tremens encountered in my wards of the Denver County Hospital. The resident interne assisting me, Dr. Elizabeth M. Collier, has continued the application of the method in such subjects since the lapse of my term of service, and reports very favorable results from it.

I have used saline infusions in four cases of scarlet fever, two of them very severe, and the results were admirable. The casts and albumin found in the urine early in the disease disappeared before the patients were ready to leave their beds.

In functional disorders marked by acetonuria, great gastric irritability. and depression of renal activity, proctoclysis and the administration of bicarbonate and salicylate of soda by rectal infusion have proved of great value.

In a number of cases of chronic pulmonary tuberculosis having a daily rise of temperature, I have failed to see decided benefit from proctoclysis. But in intercurrent febrile, "grippal" attacks the comfort of the patient has been greatly increased and the invasion apparently cut short by the procedure. In the sudden flooding of the system with toxins from confined pus, which not rarely occurs in tuberculous subjects, remarkable amelioration of the symptoms may follow a saline infusion by the bowel.

In conclusion, it must be admitted that the favorable results accompanying the use of proctoclysis in appropriate medical cases indicate that this method of *quasi* internal hydrotherapy deserves such a thoroughly detailed study as will accurately establish its indications and limitations in medical therapeutics.

[2] Denver Medical Times and Utah Medical Journal, July, 1909.
[3] Jour. Amer. Med. Assoc., January 29, 1910.

THE X-RAY TREATMENT OF STATUS LYMPHATICUS, WITH INFERENCES DRAWN THEREFROM CONCERNING THE PHYSIOLOGY OF THE THYMUS GLAND.[1]

By B. K. Rachford, M.D.

PROFESSOR OF PEDIATRICS IN THE UNIVERSITY OF CINCINNATI; PEDIATRICIAN TO THE CINCINNATI HOSPITAL, THE JEWISH HOSPITAL, AND THE GOOD SAMARITAN HOSPITAL, CINCINNATI.

In 1907 Alfred Friedlander[2] reported a case of status lymphaticus with marked enlargement of the thymus and persistent stridor, which he treated successfully by the *x*-rays. Heinecke (quoted by Friedlander) demonstrated the selective action of the Röntgen rays on lymphoid tissues, including the thymus, and showed that under their action marked changes occurred in these tissues, with a reduction in their size. Friedlander's successful case directed attention to the *x*-ray treatment of enlarged thymus in connection with status lymphaticus, and led to the successful treatment by this method of a number of other cases. The purpose of this paper is to call attention to two of these cases which came under my direct observation, and to draw therefrom certain inferences concerning the physiology of the thymus gland.[3]

Case I.—John X., born July 2, 1908, birth weight 6½ pounds, was apparently normal for one month, and was then weaned because of indigestion. When one month of age he choked on a calomel and soda powder, became greatly cyanosed, and was prostrated for a number of hours afterward. From this time on his mother noticed that there was great difficulty in keeping his feet warm, and that at times they were blue. About the middle of December, when five and one-half months old, he had an afebrile attack of "bronchitis" of the larger tubes, characterized by cough, coarse rales, wheezing, and dyspnœa; this attack lasted one month. In the middle of February he developed another attack of bronchitis, with the same symptoms as in the preceding attack, except that the cough was more violent and spasmodic. This attack continued until, on March 4, his adenoids were removed under chloroform anesthesia, in the hope of relieving these symptoms. Following the operation the child was better for two days, when the whole symptom group recurred; at this time there was marked stridor and dyspnœa, which interfered with his taking food. On April 3 a second and more radical operation was made for the removal of the adenoids and tonsils. Following

[1] Read by title at a meeting of the Association of American Physicians, Washington, D. C., May 4, 1910.

[2] Archives of Pediatrics, July, 1907.

[3] For many of the facts used in this paper I am indebted to the writings of A. S. Warthin, in Osler's System of Medicine, the Transactions of the Association of American Physicians, 1906, and the Archives of Pediatrics, August, 1909; and to Pappenheimer's paper on the Histology of the Thymus Gland, in the Journal of Medical Research, February, 1910.

this operation for two or three days there was a slight improvement in his symptoms, and then his condition grew steadily worse. The cyanosis, stridor, and prostration increased until on April 12 he was brought to Cincinnati with the diagnosis of thymic asthma. I saw him on his arrival, and asked Drs. Forchheimer and Friedlander to see the case with me. We concurred in the opinion that the child had status lymphaticus and was suffering from thymic asthma, The thymus gland, greatly enlarged, was readily mapped out by percussion. A blood examination by Friedlander showed a chloro-anemia and a well-marked lymphocytosis. The patient was pale, fat, and flabby. He suffered constantly from stridor, so severe that it was almost impossible for him to take food. He also had a severe paroxysmal cough, which came on at short intervals and increased the stridor into an attack of asthma associated with such great cyanosis that his life was threatened. The child was held the greater portion of the time stomach down, across the mother's lap; in this position the stridor was less severe. The lymphatic glands were increased in size, the spleen was slightly enlarged, and, as previously noted, the adenoids and tonsils had been removed. The tuberculin skin test was negative.

So urgent were the symptoms that the *x*-ray treatment was begun on the day of his arrival in Cincinnati, April 12, 1909. At each treatment the rays were applied both anteriorly and posteriorly over the region of the thymus. The rest of the body was carefully guarded from their influence. Sixteen treatments in all were given, extending over a period of six weeks. In the beginning the time of exposure to the rays was three minutes anteriorly and three minutes posteriorly. In the later treatments the exposure was eight minutes. The treatment was interrupted during the fourth week.

Within forty-eight hours after beginning the treatment there was slight improvement in the symptoms, especially the stridor, and this improvement continued steadily throughout the eight weeks he was under treatment. During this time the thymus gland was reduced to its normal size, and the dulness posteriorly on the left under the angle of the scapula, which we thought to be due to lymphatic enlargement and pulmonary compression, also disappeared. The paroxysmal cough and stridor had been almost entirely relieved and the child had gained steadily in weight and strength, but he still appeared anemic. On May 17 he was taken to his home in Kentucky, and within two weeks thereafter his father, who is a physician, reported that the cough and stridor had entirely disappeared, and that the child was apparently well. From the time the child returned home until the present time, April 11, 1910, he has had, at intervals of about three months, three very slight attacks characterized by cough and dyspnœa, which occurred without apparent cause and which were promptly relieved by one or two mild *x*-ray treatments. Apart from this he has presented the appearance of a sturdy infant developing

along normal lines. His father wrote me on April 11: "It will be just one year ago tomorrow since I took my baby to see you, and he now seems to be perfectly well. About three months ago he had another slight recurrence, which yielded promptly to two mild exposures to the *x*-rays. It seems that he may from time to time need an occasional treatment, but I shall remember your advice and not expose him to the rays unless there seems real occasion for it."

CASE II.—John Y., born July, 1908. When about six months of age it was noted that he had rapid heart action and difficulty in breathing from slight causes. From this time on these symptoms gradually increased in severity and the general symptom complex of status lymphaticus slowly became apparent. On November 1, 1909, I saw the patient for the first time with Dr. Sam Rothenberg, and from this time on he was cared for jointly by Dr. Rothenberg and myself. On this date the child was fifteen and one-half months old and presented well-marked and aggravated symptoms of status lymphaticus, which Dr. Rothenberg informed me had existed for some months. He was fat, flabby, anemic, and markedly lacking in physical development, especially his lower extremities. He had great dyspnœa, with well-marked stridor, which at times during the last few months was greatly aggravated by such slight causes as fits of anger, indigestion, and catarrhal conditions of the throat and nose. The child at times had a spasmodic cough, which would aggravate the stridor and produce cyanosis. The heart was dilated and its muscle weak and irritable. The action of the heart was rapid at all times, and this rapidity was greatly increased by the same causes which aggravated the stridor. The spleen, lymph nodes, and tonsils were enlarged, and there was a marked itching of the skin of the face and head, which caused the child to mutilate the skin of these regions by scratching. A well-marked enlargement of the thymus was demonstrated by percussion, and a blood examination by Dr. Max Dreyfoos demonstrated a very pronounced chloro-anemia and well-marked lymphocytosis. The blood examination was as follows: Hemoglobin, 65 to 70 per cent.; red corpuscles, fresh preparation, showed considerable poikilocytosis, a few "ameboid" micropoikilocytes, and, on the whole, the red cells were smaller than normal; the stained preparation showed 6 normoblasts to 500 whites counted; white corpuscles, 24,600; red corpuscles, 5,881,250; color index, 0.58; differential count, polymorphonuclear neutrophiles, 25.6 per cent. small lymphocytes, 61.8 per cent.; large lymphocytes, 2.6 per cent.; large mononuclear leukocytes, 8.4 per cent.; eosinophiles, 0.6 per cent.; mast cells, 1 per cent.

The patient was immediately referred to Dr. Sidney Lang for *x*-ray treatment. In all, twelve treatments were given during the next six weeks, and during this time the symptoms of status lymphaticus gradually improved. There was an early and marked diminution in the size of the thymus and marked improvement in the stridor and

heart action; the spleen and lymph nodes grew smaller and the itching of the skin largely disappeared, but the child still presented a very anemic appearance. A blood examination on December 26, 1909, showed that the lymphocytosis had disappeared, but that the chloroanemia was slightly more aggravated than before. The blood state at this time was as follows: Hemoglobin, 50 to 55 per cent.; fresh preparation, reds pale and smaller than normal; considerable poikilocytosis and anisocytosis; stained preparation, polychromatophilia; 2 nucleated reds to 300 whites counted; white corpuscles, 6,800; red corpuscles, 4,675,000; color index, 0.56; differential count, polymorphonuclears, 38 per cent.; small lymphocytes, 46 per cent.; large lymphocytes, 6 per cent.; large mononuclear leukocytes, 7.67 per cent.; eosinophiles 1.66 per cent.; mast cells 0.67 per cent.

Following the above blood examination the *x*-ray treatment was discontinued and the child was given citrate of iron hypodermically. He continued to improve in general health and the slight amount of dyspnœa which still appeared at times continued to grow less. The heart was gradually reduced in size and its action became more normal. This improvement continued until January 12, when the child was taken ill with bronchopneumonia, from which he made a rapid recovery. February 27, a blood examination was made, with the following results: Hemoglobin, 90 per cent.; fresh preparation, reds smaller than normal; moderate poikilocytosis; considerable anisocytosis; rouleaux formation fairly well marked; white corpuscles, 14,500; red corpuscles, 5,906,250; differential count, polymorphonuclears, 69.25 per cent.; small lymphocytes, 22.5 per cent.; large lymphocytes, 3.25 per cent.; large mononuclears, 3.25 per cent.; eosinophiles, 1.5 per cent.; mast cells, 0.25 per cent.

It will be noted that the special point of interest in the above blood picture is the appearance of a polymorphonuclear leukocytosis, while the lymphocyte count remains low. It was suggested by Dr. Dreyfoos that this might be due to a slight influenza which the baby had at the time, and which had developed after Dr. Rothenberg and I had seen the baby and asked for the blood count on February 20. In this connection, however, it may be interesting to note that Rudberg, quoted by Pappenheimer, notes in his study of the regeneration of the thymus in rabbits following Röntgenization, that during the later stages of this regeneration there is a marked invasion of the thymus gland with polynuclear leukocytes derived from the blood. This would indicate that a polynuclear leukocytosis might be a physiological reaction following the *x*-ray treatment of the thymus. Without other treatment than fresh air and careful feeding this child continued to improve in general health, until on April 5, 1910, he presented the appearance of a normal child. The dilated heart had returned almost to normal size, and none of the symptoms of status lymphaticus were present. The child at this time was gaining in weight and developing along normal lines, and the blood state

below given is almost, if not quite, normal: Hemoglobin, 75 to 80 per cent.; fresh preparation, red corpuscles slightly under normal size; small number of poikilocytes; white corpuscles, 9800; red corpuscles, 4,881,250; color index, 0.9; differential count, polymorphonuclears, 48.33 per cent.; small lymphocytes, 39.67 per cent.; large lymphocytes, 2.66 per cent.; large mononuclear leukocytes, 3.67 per cent.; eosinophiles, 5.33 per cent.; mast cells, 0.34 per cent.

A study of this case indicates that the disappearance of the lymphocytosis under the *x*-ray treatment of the thymus gland in status lymphaticus is so marked that a careful study of the blood state in this condition will give important information not only as to the efficacy of the treatment, but as to the length of time this treatment should be continued. It will perhaps be wise to discontinue treatment when the lymphocytosis disappears, even though there still be left some cough and stridor. The chloro-anemia which is not improved under the *x*-ray treatment may be aggravated if it be continued too long. Following the *x*-ray treatment in these cases, hypodermic injections of iron, with careful feeding and fresh air, may be necessary to restore the blood state to a normal condition. In both of the cases here reported slight cough and stridor were present when the *x*-ray treatments were discontinued, but disappeared entirely a few weeks later.

PHYSIOLOGY OF THE THYMUS. The histology of the thymus gland is very fully discussed by Pappenheimer.[4] It appears that while many points concerning the development and histology of this gland are still in dispute, it is rather generally conceded that it is relatively large in infancy and early childhood, but that it gradually increases in size up to the fifteenth year of life, and thereafter regressive changes slowly take place which diminish its physiological efficacy. It is also very generally believed that the thymus furnishes an internal secretion which exerts an important influence on nutritional processes, and that its period of greatest functional activity along this line is during fœtal life and early childhood; this function falls off most rapidly after the fifteenth year, but probably remains more or less active throughout life. For a review of the various inferences and suggestions which have been made concerning the physiological functions of this gland the reader is referred to the papers by Warthin, previously noted.

It is my purpose here to call attention to the following facts, which I believe have been demonstrated by the *x*-ray treatment of the enlarged thymus gland in the condition known as status lymphaticus, and based upon these facts I shall offer an hypothesis concerning the functions of this gland, which, if true, will have an important bearing upon the pathology of this disease.

In the *x*-ray treatment of status lymphaticus in infants and young children, although no portion of the body is exposed to the

[4] Journal of Medical Research, February, 1910.

influence of the rays except that which directly holds the thymus gland, we have as a result of this treatment:

1. Decrease in size of the hyperplastic thymus, with the disappearance of the cough, stridor, and asthma.

2. Decrease in size of the enlarged spleen and lymph nodes.

3. The exhaustion and general feebleness of constitution gives place to normal conditions of health and strength, and physical and intellectual growth are greatly stimulated.

4. A rapid disappearance of the marked lymphocytosis which characterizes this disease.

5. Excessive physiological action of the thymus gland is controlled. The slight return of the symptoms, stridor, cough, etc., at intervals of three or four months in one of my cases, and the quick control of these symptoms by one or two exposures to the *x*-rays, indicate that the gradual regeneration of the thymus following the *x*-ray treatment may be accompanied by a gradual reproduction of the same pathological conditions, hypersecretion, etc., which were present before the treatment was begun.

Since the above remarkable results are brought about by the action of the *x*-rays on the thymus gland, it would appear that the excessive physiological activity of the thymus gland bears the same relationship to status lymphaticus that excessive activity of the thyroid gland bears to exophthalmic goitre. One seems justified in inferring from the above facts that the exciting cause of true status lymphaticus acts primarily on the thymus gland, commonly producing marked hyperplasia of this organ with an increase in or perversion of its internal secretion, and that this increased or perverted secretion is responsible for the general hyperplasia of lymphoid tissues, the lymphocytosis, and general feebleness of constitution which occur in this disease. This inference seems justified by the facts above noted, that the general hyperplasia of lymphoid structures, as well as all of the other symptoms of status lymphaticus, disappear when the *x*-rays reduce the thymus to normal size and, perhaps of more importance, to normal functional activity.

If the above hypothesis be true, that under pathological conditions an excess or perversion of the internal secretion of the thymns may produce hyperplasia of the thymus gland itself and of lymphoid tissues in general throughout the body, the inference seems plain that the normal thymus gland, which is at the height of its functional development in intra-uterine and early extra-uterine life, exercises a preponderating or controlling influence over the lymphoid tissues of the body, their functional capacity being increased or diminished by the increase or diminution of the internal secretion of the thymus. This hypothesis is in accord with the increased activity of lymphoid tissues during early life, and with the gradually diminishing activity of these tissues in later life, as the physiological atrophy of the thymus gland gradually diminishes its internal secretion.

The hypothesis that the thymus gland during infancy exercises an influence over the functions of lymphoid tissues explains many of the observations which have been made in connection with this gland.

Up to the present time, according to Warthin, seven cases of enlarged thymus producing thymic asthma have been cured by the partial or complete removal of the enlarged gland. One of these cases operated on by König was an infant only nine weeks old. Complete relief from the symptoms followed the operation, but König subsequently advised against complete thymectomy in these cases, because the patient operated on by him afterward developed rickets and did not walk until after it was four years of age. It will be noted that this case was operated upon at the very time in the life of the infant when the thymus gland is most active, and the infant was thereby deprived during the first and second year of its life of the physiological function of this organ, and thereby suffered profound nutritional disturbances.

The experiments of Basch, to the effect that extirpation of the thymus in "young" dogs is followed by lack of bony development and serious nutritional disturbances, is in accord with König's observations. Stokes, Ruhräh, Rohrer, Dudgeon, Bovaird, Nicholl, and many others have noted the fact that primary marasmus is associated with thymic atrophy. Warthin says that in primary marasmus thymic atrophy appears to be constant, and that similar changes also occur in the spleen and lymph glands, "suggesting a general lymphoid exhaustion." In these cases it would appear that the thymic atrophy is the initial lesion and one of the causative factors in the production of infantile marasmus. This condition would naturally occur if by destroying the functional activity of the thymus a "general lymphoid exhaustion," or general functional debility of all the lymphatic tissues resulted, in conformity with the hypothesis above outlined.

Warthin says that "in true acephalic monsters the thymus may be entirely absent, and up to the present time such cases are the only ones in which a total absence of the organ has been definitely shown to exist." These facts apparently indicate that there is some relationship between the absence of the thymus and the lack of development here referred to. If this be true, it points to the conclusion that the thymus gland during intra-uterine life exercises, by means of some internal secretion, a powerful influence over the metabolic processes which are necessary to the normal development of the fœtus. This conclusion is in accord with the hypothesis that the thymus gland exercises a controlling influence over the development and functional activity of lymphoid tissues.

Svehla[5] prepared fresh and dried thymus glands and fed them to dogs, with the following results: (1) Drop in blood pressure, as a result

[5] Wien. med. Blatt., 1896.

of weakness and paralysis of the vasoconstrictors. (2) Acceleration of the pulse due to the action of the toxin on the heart. (3) Large doses produced death of the animals, with restlessness, collapse, and dyspnœa, and postmortem examination showed death from suffocation.

Sachsa[6] injected a 10 per cent. watery solution into crural veins, and produced a fall in blood pressure, acceleration of the pulse, and death from acute suffocation. This toxic action was more marked in young animals. Both Svehla's and Sachsa's conclusions, that thymic asthma and thymic death may be due to hyperthymization of the blood, are in accord with my hypothesis.

On finishing this paper there came to my notice the research of Lucien and Parisot.[7] These observers made a careful study of the influence of thymectomy in rabbits, and found that in very young animals removal of the thymus was followed by a noticeable retardation of growth. The changes in the skeleton were in the weight and general dimensions of bones; no curvatures, epiphyseal enlargements, or bony softenings were found. The testicles and ovaries were diminished in size, corresponding with the lack of development of the organism in general. All of these changes were less noticeable in older animals, and did not occur at all in animals that had attained their full development. These findings of Lucien and Parisot may be explained by the hypothesis above advanced.

A CASE OF SPLENOMEGALY ASSOCIATED WITH MARKED ANEMIA OF THE PERNICIOUS TYPE.

By R. D. Rudolf, M.D. (Edin.), F.R.C.P. (Lond.),

PROFESSOR OF THERAPEUTICS IN THE UNIVERSITY OF TORONTO; SENIOR ASSISTANT PHYSICIAN TO THE TORONTO GENERAL HOSPITAL,

AND

C. E. C. Cole, B.A., M.B. (Tor.),

DEMONSTRATOR OF THERAPEUTICS IN THE UNIVERSITY OF TORONTO, TORONTO, CANADA.

The following case seems worthy of being put on record, as it presents many unusual features. The patient has been under observation in London and Toronto during many years, and the history can thus be followed out in a way that is often not possible. The diagnosis offers many difficulties, but if, as seems probable, it be one of pernicious anemia, with a very large spleen, then in several ways it is a case of great rarity.

W. P., aged twenty-eight years and six months. Single. Lead glass worker. Admitted to the Toronto General Hospital on June 12, 1909.

[6] Centralbl. f. allg. Path. u. path. Anat., 1897.
[7] Archiv. méd. exper. and d'anat. path., January, 1910.

Complaints. Weakness, shortness of breath on exertion, and swelling of the abdomen.

Duration. About four weeks, but, as the history will show, he has not been really well for at least twenty years.

Family History. Father alive and well. Rheumatism four times. Mother alive but delicate. Brothers, two; one died of whooping-cough; one healthy. Sisters, none. No history of tuberculosis or syphilis in the family. No history of any disease resembling what the patient now suffers from.

Personal History. Born in London, England. Came to Canada many years ago. Has been a lead glass worker for years. Fairly comfortable surroundings and always has good food. No venereal history.

Previous Health. Was a small, weakly baby. Had measles at the age of four. When eight years old was in the Great Ormond Street Hospital suffering from "jaundice," and remained there four months. (Notes of this stay are not obtainable.)

At ten years of age was readmitted to the Great Ormond Street Hospital, March 5, 1890, complaining of headache and dull abdominal pain. A summary of his condition then, as given by Mr. R. S. Frew, the Registrar to the hospital, is as follows: "Very thin boy. Skin pale lemon color. Conjunctivæ slightly tinged. No eruptions or œdema. Lips pale and gums slightly swollen. Tongue pale. Cervical glands enlarged, especially on the left side. Temperature, 101°. Abdomen not distended. Prominence in left hypochondrium reaching to umbilicus and descending on respiration. Spleen reaches to within 1½ inches of left anterior superior iliac spine. Notch in its anterior border evident. Liver palpable below costal margin. Heart not enlarged, but systolic murmurs in all areas, conducted into axilla. Chest normal. Blood: Red cells, 1,000,000, white cells, 18,000, hemoglobin, 12 per cent.; March 25, hemoglobin, 25 per cent.; April 1, red cells, 1,625,000, white cells, 26,625, hemoglobin, 25 per cent.; April 17, red cells, 1,100,000, white cells, 7750, hemoglobin, 40 per cent.; April 28, red cells, 1,700,000, white cells, 10,000, hemoglobin, 40 per cent. Blood smear normal. Diagnosis, enlarged spleen."

Was readmitted in September, 1890 (aged ten years and nine months). "Since discharge had gradually become short of breath; lost color; had pains in head and abdomen; no hemorrhages nor vomiting. He was yellowish in color. Temperature, 100°. Glands enlarged along posterior border of sternomastoid, and shotty in left groin. Elsewhere were normal. Spleen was much enlarged. Liver not palpable. Systolic murmurs as before. Urine contained no albumin or sugar. Blood: Red cells, 1,111,000; white cells, 10,000; hemoglobin, 25 per cent.: September 17, red cells, 2,900,000; white cells, 10,000; hemoglobin, 46 per cent.: October 13, red cells, 3,500,000; white cells, 14,000; hemoglobin, 70 per cent. Optic disks normal. Sent to Convalescent Home."

February 9, 1891. "Reported at the out-patient department. Is paler again. No pain. Spleen reaches to one inch beyond the umbilicus. Blood: Red cells, 700,000; white cells, 21,200; hemoglobin, 19 per cent. After that was twice at the Great Ormond Street Hospital as an in-patient at short intervals. The condition was much the same, and he always temporarily improved."

The patient next turned up at St. Mary's Hospital, under the care of Dr. (now Sir) John F. H. Broadbent, who writes that he well remembers the case; and we are indebted to Dr. M. Mitchell Bird, the medical superintendent of the hospital for further notes, which are condensed to the following: The patient was admitted on July 23, 1894, then aged fourteen years. He complained of dyspnœa, anemia, slight cough, and extreme weakness. He stated that his periods of good health had been longer after each course of treatment at the Great Ormond Street Hospital. Had been fairly well before the present attack. He was of a pale yellow color. Spleen much enlarged. Liver palpable below the costal margin. Loud systolic murmurs audible all over precordium. Blood: Red cells, 800,000; white cells, not increased; hemoglobin, 25 per cent. He was treated with rest, arsenic, and iron, and left the hospital much improved.

During the next fourteen years of his life the patient says that he has been well and has never been laid up nor required medical advice. About a month ago, however, his old symptoms of weakness, dyspnœa, and yellow pallor returned, and he has been very poorly since. A week ago he fainted. He entered the Toronto General Hospital on June 4, 1909.

Condition on Admission. Patient is a well-developed man. Skin has a yellow and pallid hue and the cunjunctivæ are distinctly jaundiced. In the folds of the axillæ and elsewhere there is considerable pigmentation. He gives a negative Moro reaction. Temperature, 100.3°, and all through his stay in the hospital there was slight elevation of the temperature. The lymphatic glands on the right side of the body are slightly enlarged. (The enlargement was on the *left* side in boyhood according to the records.) There is no tenderness over the long bones. No wrist-drop or trophic changes suggestive of lead poisoning.

Alimentary System. The teeth are poor and carious. The spleen is greatly enlarged and reaches to half an inch beyond the umbilicus. It is firm and free from tenderness. The notch is very evident and there is a slight shallow groove across the surface of the organ. The liver is not enlarged. The stools are somewhat clay-colored.

Circulatory System. Some dyspnœa and faintness on exertion. He can lie down in bed. No œdema or cyanosis. Pulse is regular and of medium systolic pressure, but collapses between the beats, almost suggesting a "water-hammer" one. The heart is not evi-

dently enlarged. Systolic murmurs are audible all over precordium and there is a loud *bruit du diable* in the neck. There is a slight retraction at the right apex, but otherwise the respiratory system seems normal.

Urine. Quantity normal. Dark red in color, but does not give the bile reaction with nitric acid. A trace of albumin. No indican. Many crystals of calcium oxalate, uric acid, and urates.

Nervous system normal. Nothing abnormal in *fundi oculorum.*

Blood. Shows a marked anemia of the pernicious type, in that the color index was persistently 1 or above. The results of the frequent examinations are collated in the following table:

BLOOD EXAMINATIONS.

Date.	Hemoglobin.	Red blood cells.	White blood cells.	Nucleated red blood cells.	Differential Counts.	Polynuclears.	Eosinophiles.	Basophiles.	Lymphocytes.		Normoblasts.	Megaloblasts.	Total nucleated red blood cells.
									Large.	Small.			
1909													
June 12	28	1,152,000	28,000	10,890		65.40	1.57	1.57	3.77	27.67	11.31	27.60	38.91
June 13	..	1,440,000	25,700	13,261		65.00	2.00	0.60	5.80	26.40	22.25	29.35	51.60
June 14	26	.....	23,900	9,010		63.30	1.60	0.60	7.30	27.20	17.70	20.00	37.70
June 15	28	1,520,000	18,000	5,997		63.70	3.20	1.60	4.16	27.20	16.33	17.00	33.00
June 16	31	1,750,000	14,800	5,097		58.36	4.73	1.26	5.04	36.00	.....	.....	34.44
June 17	34	1,724,000	14,000	5,250		60.00	1.90	2.52	2.52	32.70	15.40	22.10	37.50
June 18	38	1,824,000	16,000										
June 19	40	1,856,000	12,000	3,624		62.42	3.18	2.20	4.80	27.07	12.40	17.80	30.20
June 20	40	2,018,000	14,800	5,772		55.60	2.50	1.20	3.20	37.30	12.90	26.10	39.00
June 21	..	2,800,000	18,000	7,560		60.10	1.60	2.20	5.10	30.87	14.80	27.30	42.10
June 22	41	2,280,000	11,600										
June 23	45	2,180,000	12,000	3,345		61.50	3.54	1.60	3.50	30.00	8.33	19.55	27.88
June 24	45	2,264,000	14,000	2,261		70.90	1.60	1.30	4.00	22.20	5.45	10.70	16.15
June 26	45	.....	11,600	2,784		65.70	1.60	0.60	6.70	25.30	7.00	17.00	24.00
June 29	46	2,300,000	11,600	1,032		67.50	2.00	1.00	3.20	26.30	3.80	5.10	8.90
July 2	48	2,600,000	11,200										
July 6	50	2,964,000	11,300	247		62.00	3.00	....	6.50	28.50	2.50	2.00	4.50
July 9	46	2,890,000	5,500										
Nov. 28	..	.....	.....	....		64.50	4.50	1.50	3.00	26.00	0.50	1.00	1.50
1910													
Feb. 13	..	.....	.....	....		74.20	5.80	1.00	4.50	14.50	0.30	1.30	1.60

The white cells showed marked signs of immaturity or a tendency to degeneration. Some large mononuclear forms seemed closely to resemble myelocytes, but their structure was too definite. Many of the polymorphonuclears were very small, and a tendency to acid staining was evident.

The chief change in the blood was in the red cells. Fully 50 per cent. of the non-nucleated forms showed staining in an atypical bluish tint. Stippling was marked, varying from a few coarse blue granules to hundreds of coarse or fine ones, so closely packed as to produce a close resemblance to an atypical white cell. The question naturally arises here of whether the lead-working occupation of the patient had anything to do with this, but as both in pernicious anemia and in splenic anemia such staining is common,

this suspicion loses much of its weight. The poikilocytosis of the non-nucleated forms was mainly in regard to size, although many cells were irregular in outline and even rosette in form. Some of them were very large.

Many cells were so destitute of staining as to appear as mere ghosts, with just a faintly staining outline.

The nucleated red cells were exceedingly variable in form. The majority of them showed a primitive form of nucleus with a definite chromatin network and frequently more than one nucleolus. They answer very closely to the appearance of bone marrow, or lymphoid cells, which, according to many are the mother cells alike of the red and most of the white corpuscles. These cells not infrequently showed mitotic figures. The other nucleated red cells showed a uniformly intensely blue staining nucleus, or several nuclei, up to seven; an atypical budding process appearing in some cases. Free nuclei of this description were frequently noted in the smear, some just becoming detached from the cell body. These nuclei made the differentiation of lymphocytes a difficult matter. The majority of the nucleated red cells were megaloblasts, though normoblasts and some microblasts were present.

The smears were made on slides, stained by Wright's usual technique, and were counted under a Zeiss $\frac{1}{12}$ oil-immersion lens. The cells were counted by the same Thoma-Zeiss hemocytometer throughout.

The hemoglobin was estimated by two separate readings with the Dare hemoglobinometer.

Smears made by the patient at his home in November and February last showed more regularity in size and shape of the red cells, but still many nucleated forms. The color index as mentioned was always 1 or above 1.

During his stay in the hospital the patient improved greatly, he rapidly gained strength, his jaundice disappeared, and the spleen became rather smaller.

On February 13 last he writes that he thinks that his spleen is down to *its* normal size, and that he feels as well as he ever did in his life. This letter was of the same date as the last smear in the table, in which the still abnormal condition of the blood is evident.

Remarks. The blood picture alone would suggest a diagnosis of pernicious anemia; the marked number of nucleated red cells and especially the great proportion of megaloblasts making it probable that we had to deal with the plastic form of this disease. The splenomegaly, although being unusual in pernicious anemia, would by no means exclude it, as cases are on record in which the spleen was quite as big as in our case, in which the disease was undoubtedly of the pernicious type. Osler records such a case, which was at first taken to be one of splenic anemia, but subsequently came to

postmortem and proved to be one of pernicious anemia,[1] and in something like 1 per cent. of all cases of pernicious anemia the spleen is so large as to reach to the umbilicus.[2] But assuming for the moment that the case be one of pernicious anemia, even then it presents several most unusual features. In the first place the disease commenced at the age of eight years, if not earlier, which is very unusual. Again, it has lasted for at least twenty years, and the patient is still in comparatively good health; and yet the longest cases recorded, apart from six cases of apparent recovery, only lasted between fourteen and fifteen years (Cabot[3]). Further, according to the patient's history, he was so well between the ages of fourteen and twenty-eight that he was never laid up nor required any medical advice—that is, he seemed to have a remission of fourteen years, and yet the longest recorded remission from prostrating symptoms on record is six years,[4] although McPhedran[5] recently has reported a case in which there was a remission for seventeen years.

As regards splenic anemia, the enormous size of the spleen with the marked anemia and a normal differential white blood count would superficially suggest such a diagnosis, but the very profoundness of the anemia would be against this conclusion, and further, the fact that the anemia was always of the pernicious form with a high color index would seem to exclude this diagnosis.

Other forms of splenomegaly with anemia, such as congenital syphilis, tumor, malaria, and even a possibility of filaria were all carefully weighed and excluded.

The case would appear to be either one of pernicious anemia of an exceptionally prolonged type, commencing at an exceptionally early age, and associated with an exceptionally large spleen, or one of splenic anemia with an exceptional blood picture.

The patient has left Canada for the United States, and we hope that his condition may be followed out by whomsoever he may consult.

[1] Transactions of the Association of American Physicians, 1902.
[2] Osler's Modern Medicine, iv, 765.
[3] Ibid., 635.
[4] Ibid., 633.
[5] Amer. Jour. Med. Sci., 1910, cxl, 261.

THE SPINAL CORD LESIONS IN TWO CASES OF PERNICIOUS (ADDISONIAN) ANEMIA.

By C. N. B. Camac, M.D.,

PROFESSOR OF CLINICAL MEDICINE IN THE CORNELL UNIVERSITY MEDICAL COLLEGE, NEW YORK,

AND

Lindsay S. Milne, M.D.,

RESIDENT PATHOLOGIST, RUSSELL SAGE INSTITUTE OF PATHOLOGY, NEW YORK.

The following cases of spinal cord lesion with anemia belong to that group first described by Lichtheim[1] in 1887, and by many observers since, such as Bastianelli[2], Burr,[3] Clarke,[4] Jacob,[5] Jacob and Moxter,[6] Lenoble,[7] Putnam and Taylor,[8] Russel,[9] Nonne,[10] and especially by Putnam[11] and Dana[12] in this country, and recently termed by certain German observers pseudocombined sclerosis. The two patients whose records are presented were under the independent observation of several clinicians in three large hospitals of New York: Drs. W. Gilman Thompson, W. B. Pritchard, L. A. Conner, and T. W. Hastings, to whom we are indebted for the early records, extending back in one case three years, in the other five years. The first case we saw in Dr. Pritchard's service in the City Hospital, and the second case was under the personal observation of one of us for a year in the Cornell Dispensary and the City Hospital.

Case I.—*Under observation five years; symptoms and signs of pernicious anemia; no neurological features; at the postmortem the characteristic changes of pernicious anemia; in the cord, from the*

[1] Zur Kenntniss der im Verlaufe der perniciosen Anämie, etc., Verhandl. des VI Congress f. innere Med., Wiesbaden, 1887, p. 84.

[2] Le sclerosi combinale de midollo spinalle nelle anemie perniciose, Bulle. Roy. Acad. di Roma, 1895-96, xii.

[3] The Spinal Cord Lesions and Symptoms of Pernicious Anemia, Univ. Med. Mag., 1895, vii, 472.

[4] Remarks on the Changes in the Spinal Cord in Two Cases of Pernicious Anemia, Brit. Med. Jour., 1897, ii, 325; On the Spinal Cord Degeneration in Anemia, Brain, 1904, xvii, 441.

[5] Rückenmarkserkrankungen bei letaler Anämie, Fortschr. d. Med., xv, 569.

[6] Uber Rückenmarkserkrankung und Veränderungen bei todlich verlaufenden Anämien, Deut. med. Woch., 1898, xxiv, 152.

[7] Contribution à l'étude des lésions médullaires dans l'anémie pernicieuse progressive protopathique, Rev. de mèd., 1897, vi, 425.

[8] Diffuse Degeneration of the Spinal Cord, Jour. Nerv. and Ment. Dis., 1901, xxviii, i, 74.

[9] The Relation of Some Forms of Combined Degeneration of the Spinal Cord to one Another and to Anemia, Lancet, 1898, ii, 4.

[10] Klinischer und anatomischer Beitrag zum Capitel der Prognose der anämischer Spinalerkrankung—2 Falle von anämischer Spinalerkrankung mit Dissociation der Anämie und des Spinalleidens, Mitteilung aus der Hamburgischer Staatsanstalten, 1907, ii, 1 to 7.

[11] A Group of Cases of System Sclerosis of the Spinal Cord Associated with Diffuse Degeneration; Occurring in Enfeebled Persons, Past Middle Life, and Especially in Women; Studied with Particular Reference to Etiology. Jour. Nerv. and Ment. Dis., 1891, xviii, 69.

[12] The Degenerative Diseases of the Spinal Cord, with the Description of a New Type, Jour. Nerv. and Ment. Dis., 1891, xviii, 205; Subacute Combined Sclerosis of the Spinal Cord and its Relation to Anemia and Toxemia, Jour. Nerv. and Ment. Dis., 1899, xxvi, 1.

tenth dorsal to the first lumbar segments on the left side there was swelling, with cavity formation, occupying almost exclusively the gray matter; patchy degeneration of the posterior columns, especially in the lower cervical region.

The patient was admitted to Bellevue Hospital, August 24, 1904, where he remained until December 13, 1904. The state of the blood during this time is shown in Fig. 1.

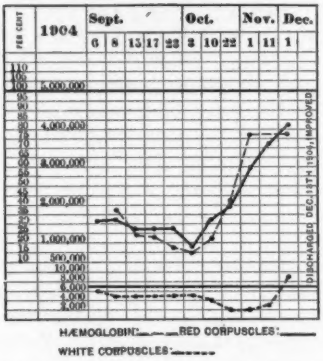


FIG. 1.—The blood chart of Case I in 1904. Microscopic examination showed the characteristic features of pernicious anemia. The highest lymphocyte count (October 3, 1904) was 37 per cent., with a polynuclear neutrophile count, on the same date, of 55.6 per cent. Normoblasts were usually present; and reached 34 in a slide on October 10, 1904. At this time 9 megaloblasts were also found, but thereafter nucleated red cells were not seen.

The following is a brief summary of the notes made on admission: A history of syphilis with initial lesion nine years previously, and excess in alcohol. Two months before admission the feet and legs swelled. Headache, dizziness on rising from recumbent position, burning pain across the abdomen, numbness in the hands and feet, and shortness of breath. Compelled on account of weakness to give up work. On examination, the diagnosis of pericarditis, chronic valvular disease, and chronic catarrhal gastritis was made, in addition to that of pernicious anemia. The stools were negative for parasites and ova. The gastric analysis showed a total acidity of 0.00073; free organic acids and salts, 0.00063; combined and free hydrochloric acid, 0.

There was nothing referable to the nervous system except persistent headache, pain over the chest and abdomen, and numbness on the left side of the body extending to the left side of the face, tongue, and lips. This latter condition occurred in paroxysms of about six minutes' duration. No objective disturbance of the nervous system was noted.

On December 13, 1904, he was discharged improved. Between

this date and January 12, when he was finally admitted to the City Hopital, he was under observation at the Metropolitan Hospital, Cornell Dispensary, and, for a brief period (one week for alcoholism) at the City Hospital. The records from these clinics show that cardiac insufficiency with valvular disease, and alcoholism with its accompanying gastro-intestinal disturbances, were the chief features. Weakness in the legs is the only note suggestive of any nervous affection. He labored in a drop-forge and coal yard and at other occupations requiring considerable muscular effort.

When admitted to the City Hospital the condition of his blood was as shown in Fig. 2.

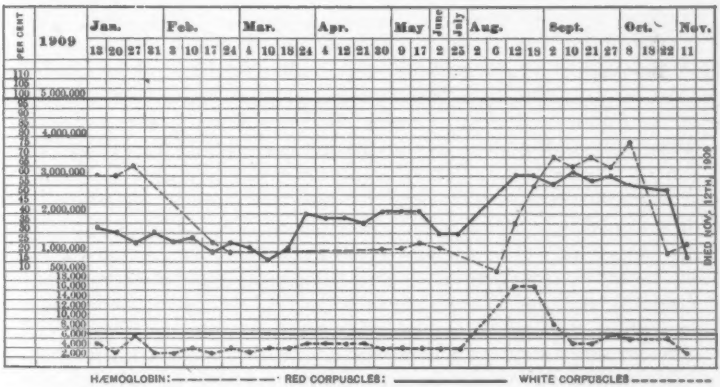


FIG. 2.—The blood chart in Case I in 1909. The microscopic examination was typical of pernicious anemia. The highest lymphocyte count was 45 per cent. (July 25, 1909); the polynuclear neutrophile count, at the same time, was 38 per cent. Nucleated red cells were a constant feature, megaloblasts preponderating.

The following is a summary of the clinical notes:

Male, aged thirty-eight years. Complains of pain in the chest, dyspnœa, dizziness, faintness, and tingling in feet and legs History of sore tongue and bleeding gums of some months' duration. Examination showed a well-nourished man, with good amount of adipose tissue; yellowish skin. The heart showed signs of chronic valvular disease with acute incompetency. The liver and spleen were palpable. Other signs were those of profound anemia. At no time were there any symptoms or signs referable to the nervous system. The pupillary reflexes were active and knee-kick was present. No paresthesias were noted. Shortly before death the patient was walking about the ward. Up to a month before his death the blood (Fig. 2) had been steadily rising, reaching nearly as high as it did five years before, when he had been able to return to heavy labor. A month prior to death, however, the erythrocyte count fell rapidly from 3,800,000 to 1,500,000. In view of the extensive

changes in the cord found postmortem, all these clinical points are of considerable significance. Intestinal parasites or ova were not found. There was absence of free and combined hydrochloric acid.

The following notes are selected from those made between July 10, 1909 and November 12, 1909, the date of death:

July 4, 1909. Has been vomiting a great deal, takes little nourishment; restless at night; dyspnœa; moderate cyanosis; heart irregular; pallor much more marked; cheeks colorless; irrational at times; asthenia extreme; sways slightly when he walks and requires assistance, but presents no peculiarity of gait; confined to bed.

July 10. Condition gradually improving; nausea and vomiting stopped; appetite returned; cyanosis disappeared; dyspnœa slight.

August 8. Improvement more marked (Fig. 2).

August 30. Improvement steady; able to walk about unassisted; marked improvement in blood (Fig. 2.)

September 22. Six or seven fluid stools daily for two days. Temperature 102°; nothing in the physical examination to account for the temperature; no blood or mucus in the stools.

September 29. Stools and temperature normal; general condition good.

October 31. Up and about ward since note of September 29, but to-day he says he has not been feeling well for ten days; weakness, dizziness; roaring in head; precordial pain; loss of appetite; skin has undergone striking change in color (yellower); returns to bed.

November 6. Is very dizzy, even in bed.

November 11. Asthenia very marked; lemon tint of skin very marked. Patient is drowsy and he gives an expiratory grunt; answers questions rationally. There is a jugular impulse synchronous with the carotid. Cardiac impulse distinctly visible and seen over a large area, but feeble to the touch. A systolic murmur to the right of the sternum different in character from that heard at the apex. Liver does not pulsate but is readily palpable. Examination of feces negative. Urine examination showed occasional hyaline casts; faint traces of albumin; specific gravity between 1012 and 1030. Examination of gastric contents made on September 8, 1909 showed no free or combined hydrochloric acid.

November 11. The patient died.

Postmortem (ten hours after death): A well developed male, rather poorly nourished and apparently about forty years of age. The skin presented a uniform lemon yellow color. The sclerotics and external mucous membranes were not pigmented. There was a considerable degree of gingivitis and the tongue was also very furred, but neither condition appeared more marked than that commonly found at autopsy in cases which have had other causes of death. The teeth were in fairly good condition. There were no scars, skin eruptions, or hemorrhages, superficially anywhere, nor

were the surface lymphatic glands or the thyroid enlarged. The left pleural cavity contained 4 ounces of clear fluid, and the right 10 ounces of a similar fluid. The subcutaneous fat was rather less than normal in amount, and the muscles were of a bright dark brownish-red color. The peritoneal cavity contained no excess of fluid, and there was no chronic peritonitis. The pericardium contained 4 ounces of clear, straw-colored fluid and showed no evidences of pericarditis.

The heart weighed 400 grams, was soft and flabby, and all its chambers were slightly dilated. The tricuspid orifice admitted four fingers and the mitral three. There was no deformity or acute endocarditis of the valves. The right side was relatively more dilated, and the myocardium of the right ventricle was slightly hypertrophied. In the myocardium of the left ventricle, just under the endocardium and particularly near the mitral orifice was a typical thrush-breast appearance from fatty degeneration.

The lungs were slightly pigmented and œdematous. They were also uniformly somewhat emphysematous. The bronchial and mediastinal lymph glands were not enlarged.

The mesenteric and peritoneal lymph glands were all slightly enlarged and of a pinkish white-color. Microscopically they showed a considerable proliferation of the lymphoid cells, many having become somewhat swollen and often containing some brown pigment.

The intestines were somewhat collapsed throughout and were rather transparent, and their lining mucosa appeared very smooth and atrophied. Microscopically the glands were considerably degenerated and in many parts disintegrated to a considerable extent from postmortem changes. In parts also there were some small aggregations of inflammatory large mononuclear cells.

The stomach was slightly dilated, its walls thin and its lining mucosa smooth and atrophic. Microscopically this mucosa showed some considerable atrophy and degeneration of the glands, but no marked inflammatory processes.

The liver weighed 1450 grams, was very soft in consistence and of a brownish-yellow color. It gave a very marked Prussian blue reaction for iron. Microscopically it showed an extensive fatty degeneration, particularly located in the inner half of the lobules, around the central veins. In these areas the liver cells were very extensively necrotic, while those at the periphery were much better preserved. No vascular thrombosis could be observed. There was also a very marked deposit of pigment granules in the liver cells, especially toward the periphery of the lobules. The greater part of this pigment gave the reaction of hemosiderin and with prolonged staining practically all the pigment gave the Prussian blue reaction. The lining endothelium of the intralobular capillaries in occasional places showed a very marked swelling, proliferation,

and specialization, the formation of blood constituents being in these places particularly easy to observe. There was also in considerable numbers, coarsely granular eosinophile cells, with all sizes and types of nuclei, but mostly small, and with one or several nuclei like those of a normoblast. These were attached to the walls of the capillary, as a rule, but also occasionally lay free.

The bile passages were clear. The gall-bladder contained a small amount of dark, inspissated bile, but no stones.

The spleen weighed 450 grams, was considerably enlarged, soft in consistence, and dark brownish-red in color, the Malpighian bodies not appearing evident. It gave grossly a slight Prussian blue iron reaction. Microscopically it showed a rather atrophic appearance of the Malpighian bodies. The sinuses all appeared somewhat distended and for the most part contained a large proportion of nucleated cells. Some of these cells resembled megaloblasts and some, less frequently, were normoblastic in type. Many of these cells had the appearance of developing locally, rather than of being collected from the general circulation. As in the liver, with considerable frequency, coarsely granular, eosinophile cells could be seen, of all shapes, but mostly with one or several small normoblastic-like nuclei.

The suprarenal glands were considerably atrophied.

The kidneys were slightly enlarged and of a pale, brownish-yellow color. They gave a very distinct and rapid iron reaction, chiefly in the cortex. Microscopically there was a very extensive fatty degeneration of the secreting tubules, and also a very considerable deposit of iron-containing pigment.

The bladder and prostate showed no special change.

The pancreas also appeared well preserved.

The bone marrow in the centre of the shaft of the femur had a uniform, dark raspberry color. Microscopically the fat cells had almost all been replaced by a new cellular development. These new cells were mostly erythroblastic, there being megaloblasts and normoblasts in very large numbers. There was also a very considerable leukoblastic change, as there was very numerous collections of faintly acidophile myelocytes in all stages of transition. There were relatively only a very few eosinophile myelocytes.

The mastoids and tympani appeared healthy.

The cerebral meninges showed no abnormality and the vessels at the base of the brain were well preserved. The brain was firm and in good condition. It was œdematous and showed no other gross lesion. Microscopically in the cortex, in several parts examined, and in the pons, no special lesion could be observed. The medulla for the most part showed no abnormalities, but several very small patches of degeneration in the posterior columns, in the extreme lower part. The nuclei of the medulla appeared well preserved.

The spinal cord presented somewhat extensive lesions. Although the autopsy was done ten hours after death, the brain and cord were removed from the body thirty-eight hours after this. Ten c.c. of 10 per cent. formalin, however, was injected in the usual site of lumbar puncture ten hours after death.

The membranes of the cord showed no abnormality nor grossly did the entering vessels or nerves. There was no excess of fluid within the membranes, discounting the small amount of formalin injected. The cord, with the exception of a small portion, in its entire course showed no gross change, there being no shrinking, deformity or softening, and was in a good state of preservation. This exception was between the level of the tenth dorsal and first lumbar segments where, for a distance of about 2 cm., it was very

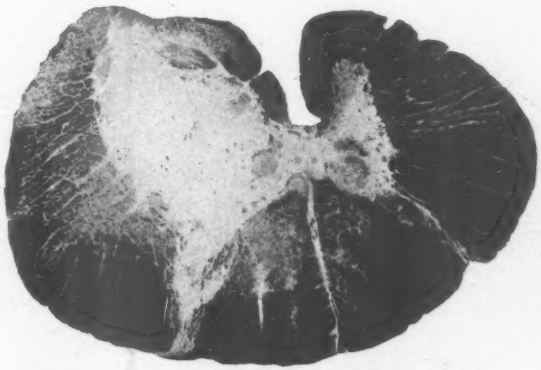


FIG. 3.—The spinal cord in Case I, showing extensive destruction of the gray matter of the left side.

much swollen on the left side. The right side, opposite this, did not appear to be affected. There was no communication externally from the interior of this swelling and no discoloration or meningeal change over it. The vessels, however, were somewhat congested on the surface of this part. On section of this swollen area, there could be seen a very extensive destruction of the cord. A large space containing a clear fluid occupied a large part of the left side in this position. This space was not surrounded by any noticeable inflammatory reaction or sclerosis, yet it was rather well defined. It occupied almost exclusively the gray matter, and the process extended between the tenth dorsal and the first lumbar segments. In the upper and lower parts there was only a very small aperture in the gray matter, on the left side, and in the deeper part of the anterior horn. This cavity formation became gradually larger until it occupied a very large part of the gray matter on the left side.

In the middle portion, opposite the twelfth dorsal segment, the gray matter was completely destroyed and the cavity extended out and laterally almost to the surface of the cord (Fig. 3). In all its length this process was located chiefly in the anterior horn, which, as the cavity became larger, destroyed it almost completely, before the posterior horn was much affected, and even when the cavity was relatively very large, the posterior part of the posterior horn largely escaped damage (Fig. 4). In all situations this lesion caused, according the to size of the cavity, a swelling of the gray matter on the left side, particularly of the anterior horn. Where the cavity grossly could not be seen, as in the upper and lower extremities of this process, the gray matter on the left side, particuliarly anteriorly, appeared swollen (Fig. 5).

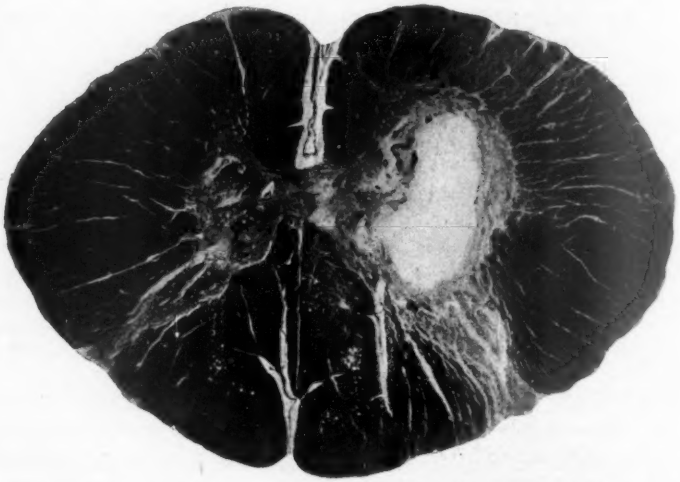


FIG. 4.—The spinal cord in Case I, showing destruction and cavity formation in the gray matter of the left side. (The specimen has been reversed.)

Microscopically there was a patchy degeneration in the posterior columns. In the lower cervical regions it affected irregularly a large portion of both Goll's and Burdach's tracts (Fig. 6). In the dorsal region the degeneration was not nearly so extensive, but there were patches in the interior of the posterior columns in the entire course of the dorsal cord and upper lumbar segments, becoming less extensive from above downward. In the sacral and lower lumbar regions, no degeneration existed. In these areas there was a disintegration of the nerve fibrils, and also a considerable amount of replacement gliosis. There were numerous large vacuoles, sometimes containing remnants of degenerated nerve fibrils. In these

same degenerate areas, however, always some healthy nerve fibrils remained and some also showing a homogeneous swelling of the myelin sheath, very often associated with disappearance or disintegration of the axon. There were, however, no evidences of inflam-

FIG. 5.—The spinal cord in Case I, showing swelling of the gray matter of the left side from œdematous separation of the tissues.

FIG. 6.—The spinal cord in Case I, showing patchy degeneration of the posterior columns.

matory reaction. The other columns of the cord showed no degenerative changes. The ganglion cells in the gray matter showed no particular change beyond perhaps a slight excess of pigment granules. In the area of extensive destruction seen grossly, the tissues seemed

more split up and distorted than necrotic. In those parts where the aperture appeared small in the gross, the tissues were simply separated apparently by fluid. There was very slight or no inflammatory reaction around the cavity. There were occasionally a few leukocytes, but no hemorrhage around or in the disintegrated part. The cavity contained only some clumps of granular debris. In the greater part of this lesion, where the cavity formation was small, the nerve cells and tissues of the gray matter were simply separated. Where the cavity was largest, the nerve cells of the entire anterior horn were largely destroyed, but this was only for

FIG. 7.—The spinal cord in Case I, showing the microscopic appearances of the gray matter in the section shown in Fig. 6. The small veins are surrounded by distended perivascular spaces and one of them is filled by a recent thrombus.

a short distance, and even in this place the cells of Clark's columns and of the posterior root were comparatively unaffected. All the vessels in the affected area on the left side were surrounded by a wide space, as if from œdema, and several of the smaller capillaries and veins, especially just above where the cavity was largest, showed recent thrombi (Fig. 7). The pial vessels in the white matter and the vessels in the gray matter at the same levels on the right side did not show any congestion or œdema. In this case, although postmortem processes may have to some extent influenced the appearances presented, yet it seemed to be the result of thrombosis of some of the small branches of the anterior spinal veins, with resulting effusion of fluid, with separation of the tissues, and some mechanical destruction of the related structures in the gray matter.

CASE II.—*Under observation three years; symptoms and signs of pernicious anemia; headache; dizziness; tingling and numbness in the hands and feet for two and one-half years; six months before death there was spastic paraplegia and paresthesia; at the postmortem the characteristic changes of pernicious anemia; degeneration of the posterior and lateral columns of the spinal cord; extensive degeneration of the posterior columns and crossed pyramidal tracts, chiefly from the midcervical to the lower dorsal regions; slight degeneration of the direct pyramidal tracts.*

Case II presented the symptoms and signs of pseudocombined sclerosis (Henneberg). The nervous manifestations were more or less present during the three years he was under observation, but when admitted to the New York Hospital, the diagnosis of pernicious anemia was recorded as the primary disease, and he was assigned

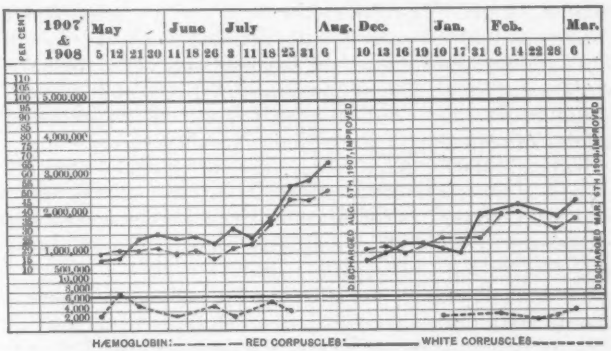


FIG. 8.—The blood chart of Case II. The microscopic examination was typical of pernicious anemia. The highest lymphocyte count was 58 per cent. (July 18); the polynuclear neutrophile count on the same date was 25 per cent. A striking feature was the absence, for the most part, of megaloblasts. Normoblasts were constantly observed, although not in great numbers.

to the Medical Division, while two and a half years later, when admitted to the City Hospital, the case was regarded as primarily neurological, and he was assigned to that division of the hospital. The postmortem examination revealed the characteristic changes of pernicious anemia, and in consequence his early record was traced.

He was admitted to the New York Hospital, May, 1907. He denied having had syphilis, but gave a history of alcohol in excess up to three or four months previously.

Fig. 8 shows the blood condition on his two sojourns in the hospitals, from May, 1907 to March, 1908. There was slight fever, the temperature ranging between 100° and 101°. There were no marked disturbances of the nervous system found during this

period. The following clinical notes are selected as indicating the condition of his nervous system at this time.

December 10, 1907. Knee-jerks not obtained.

January 7, 1908. Pain in chest, headache, tingling and numbness in hands and feet.

February 11, 1908. Taking short walks on the street.

On leaving the hospital he was able to work (hotel clerk) for three months, but had to give up on account of weakness, and a few months later he developed cramps in both legs. He was not confined to bed but sat at home, the weakness in the legs steadily increasing. This weakness continued to increase until August, 1909, when, on attempting to walk, he fell repeatedly.

In September, 1909, he was admitted to the City Hospital. On admission he was found unable to walk. Well-developed male, aged fifty-five years. Skin is lemon-color. Eyes negative; upper extremities normal. The lower extremities give exaggerated reflexes with spasticity. Skin hyperesthetic. Sensation to heat is lost below crest of ilium with exception of the inner surfaces of the thighs. The blood shows oligocythemia and nucleated red cells and anisocytosis.

November 23, 1909. Patient is drowsy and incontinent of urine. Complains of abdominal pain. Mental condition good.

December 1, 1909. Knee reflexes absent. Tactile sense greatly diminished in the lower limbs. Muscular atrophy marked and there is great loss of power. Subcutaneous œdema and numerous vesicles have appeared over both legs, and bedsores over hips and sacrum.

December 5, 1909. The patient died.

Postmortem (fifty-one hours after death): A well-nourished man, apparently sixty years of age. There was no œdema, scars, skin eruption, or hemorrhages. The skin was uniformly of a light lemon-yellow tint. The superficial lymphatic glands and thyroid were not enlarged. There was a moderate degree of gingivitis, and the teeth were considerably decayed. The superficial fat was found to be in considerable amount, and the skeletal muscles were of a dark brownish-red color. Both pleuræ were somewhat adherent posteriorly, but there was no fluid on either side. The abdomen showed no ascites or peritoneal adhesions. The pericardium contained a few cubic centimeters of a straw-colored fluid, but showed no evidence of pericarditis.

The heart weighed 425 grams, was very soft and flabby, and slightly dilated in all its chambers, particularly on the right side. There was in this case no thrush-breast appearance from fatty degeneration in the left ventricle, but the myocardium of the whole heart was evidently very considerably degenerated. The aorta and larger vessels were very well preserved. The lungs were œdematous and very emphysematous. The mediastinal, bronchial, mesenteric, and retroperitoneal lymph glands appeared pale white on section, and did not show any special enlargement.

The intestines were somewhat dilated and apparently rather

atrophic. Microscopically a very marked change was evident. The lymphoid elements were markedly hyperplastic and there were also evidences of extensive chronic inflammatory processes. The glands were largely degenerated and in many places had disappeared, their places being taken by young fibrous tissue or aggregations of large, frequently phagocytic, mononuclear cells. There was practically no pigment deposit in this situation.

The stomach was slightly dilated and its mucosa smooth and atrophic-looking. Microscopically there was considerable atrophy and degeneration of the glands, in parts also, small hyperplastic lymphoid aggregations, and also widespread subacute inflammatory processes.

The liver weighed 1600 grams, was very friable, and of a dark brownish color. It gave a very rapid and very pronounced Prussian blue iron reaction. Microscopically it showed a very widespread fatty degeneration, not being particularly localized in the central parts of the lobules, as it was in Case I. There was also a very considerable deposit of iron-containing pigment in the liver cells throughout the lobules, and also in all the tissues of the organ, including the lining cells of the vessels. In certain places the lining cells of the capillaries in the lobules were swollen, forming small groups, but no definite appearances of reformation of blood cells were observed. The gall-bladder contained a small amount of orange-colored bile, but no stones. The bile passages were free.

The spleen weighed 325 grams. It was slightly enlarged, very soft, and of a dull red color. The Malpighian bodies were not evident. It also gave a very pronounced Prussian blue iron reaction. Microscopically the Malpighian bodies were large and irregularly defined, evidently somewhat hyperplastic. All the sinuses appeared to be filled with nucleated cells. These cells were very frequently the seat of a deposit of hemosiderin. Frequently some small clumps of nucleated, hemoglobin-containing red cells of normoblastic, but more frequently of megaloblastic type, could be seen adhering to the walls of the sinuses. It seems as if there were, then, some little regeneration of blood cells taking place in these sinuses from endothelial proliferation and specialization.

The kidneys were slightly enlarged, pale, flabby, and of a pale yellow, slightly brownish color. They gave typically in their cortical parts a very bright Prussian blue reaction. Microscopically there was a very extensive fatty degeneration involving particularly the cells of the secreting tubules.

In none of these organs was there any amyloid change. The bladder and prostate were negative. The pancreas also showed no abnormalities, except slight atrophy.

The bone marrow in the centre of the shaft of the femur was dark red in color. Microscopically the erythroblastic and leukoblastic change was not so marked as in the first case, yet very largely the

fat spaces had been replaced by aggregations of regenerating blood cells. There were in these places numerous megaloblasts and normoblasts, also frequent myelocytes in all stages of transition to polymorphonuclear cells. In this case also coarsely eosinophile cells could only very occasionally be observed.

The mastoids showed no signs of suppuration.

The large vessels at the base of the brain were very well preserved and the membranes showed no abnormalities, grossly or microscopically. The brain was slightly œdematous. It showed on minute examination no gross lesion. Microscopically the cortex of the Rolandic and frontal areas, in the pieces examined, showed no special change and the internal capsule, crura, and pons, likewise.

FIG. 9. The spinal cord in Case II, showing combined degeneration of the posterior and lateral tracts; the direct pyramidal tracts are also considerably degenerated.

In the lower part of the medulla there was a slight patchy degeneration in the lateral and posterior tracts. In the region of the decussation of the pyramids, it existed only in irregular streaks, and above this no change could be seen. The nuclei of the medulla were not especially degenerated.

The membranes of the cord were in good condition, as also were the nerves and vessels relating to it. There was no excess of fluid in the spaces surrounding the cord. The cord itself was firm, and in a fairly good state of preservation. Grossly it showed no deformity or local shrinkage or softening, but there was on section a slightly darker appearance than normal of the posterior and lateral columns.

Microscopically it was evident that throughout practically the entire length of the cord there was a very extensive degeneration of the posterior and lateral columns (Fig. 9). This degeneration was most extensive in the lower cervical region, becoming less marked above this, and gradually also below it. In the sacral region, it was only very slightly evident after the first segment. In all situations it was irregular, not being confined to any well circumscribed area, and not strictly adhering to the cord tracts. The posterior column lesion was rather better defined, and apparently of older standing than was the change in the lateral columns; and the lateral column lesion became less extensive and less complete in the lumbar region than the corresponding change in the posterior columns. There were, however, degenerative changes noticeable in the lateral columns as low as the lower lumbar region. Throughout the entire length of the degeneration in the posterior columns, and especailly in the dorsal region, the outer part of the column of Burdach, for a narrow strip along the posterior tract, was comparatively unaffected. The degeneration in the lateral columns did not exclusively affect the crossed pyramidal tract. The direct cerebellar and adjacent parts of Gowers' tract were generally irregularly involved. The direct, or anterior, pyramidal tract was very irregularly affected, being degenerated for short areas on one or both sides.

The degeneration in all these situations, which was characteristically irregular, appeared as a disintegration of the nerve fibers with solution of the myelin sheaths. There was thus produced a vacuolated, somewhat reticulated, appearance. Even in the most degenerated areas some unaffected nerve fibrils remained, and many fibrils also had the appearance of a solid, homogeneous body, containing no central axon. There seemed to be no inflammatory reaction in these areas and relatively only a small amount of replacement gliosis. There were apparently no hemorrhages or inflammatory changes anywhere. The gray matter was intact throughout. Many of the ganglion cells showed some slight evidence of degeneration, probably postmortem in origin, and often were somewhat overpigmented, but there were no evidences of inflammation or of hemorrhage.

The vessels of the cord in general, and in the degenerated areas showed no particular abnormality, although several of the small arteries did occasionally show a slight degree of hyaline degeneration. In no situation was any process of endarteritis found.

These two cases illustrate two of the types of nervous-system involvement occurring in the course of pernicious anemia. In the first case, which had the longest and more pronounced history of anemia, the nervous symptoms were at a minimum and the posterior columns of the cord, particularly in the cervical region, alone

showed degeneration, characteristically patchy in distribution. In the second case the nervous involvement, particularly in the later stages, overshadowed the anemia. Here the spinal cord presented very extensive, yet incomplete degeneration with slight replacement gliosis in the posterior columns, and also a similarly irregular but more diffuse degeneration in the lateral tracts, which, however, was a rather less complete and apparently somewhat more recent, process.

The nervous manifestations observed clinically in both cases did not quite correspond in severity with the extent of the pathological spinal cord lesions, a fact which has been repeatedly noted (Strümpell,[13] Dinkler,[14] etc). It bears repeating, however, that cases of combined sclerosis pass the neurologist without the gravity of the anemia which sometimes accompanies it, being appreciated. It is also true that the internist overlooks the fact that in pernicious anemia changes may be taking place in the cord, which only delicate tests by a skilled neurologist can detect. Most of the records bear out this statement.

The occurrence of changes in the nervous system, particularly what has been termed pseudocombined sclerosis, associated with pernicious anemia, has been very frequently and extensively described since the time of Lichtheim, in 1887, and has thus come to be a well recognized possible event. Such lesions in the spinal cord in pernicious anemia have been described by practically all observers, as an irregular degeneration or sclerosis in the posterior columns, with, in many cases, a similar associated process in the lateral, particularly pyramidal, tracts. Kast[15] and Alt,[16] however, have described local changes in the medulla in leukemia, and Dana and others have noted in the spinal cord localized areas of softening from vascular obstruction. The clinical symptom complex now termed pseudocombined sclerosis is well reviewed in the publications of Collier, Batten, and Russell,[17] in 1909, and Siemerling,[18] in 1909.

This combined tract degeneration as the advanced stage of nervous involvement and, at least, degenerative changes in the posterior columns, have been noted with identical appearance in a great variety of chronic, toxic, cachectic, or anemic conditions. Dana gives 10 per cent. as the number of cases described as combined sclerosis which were due to pernicious anemia. Of Taylor's[19] 50 cases, 6 had pernicious anemia. Besides this there have been

[13] Ueber eine bestimmte Form der primären combinirten Systemerkrankung des Rückenmarks, Arch. f. Psychiatrie, 1886, xvii, 217.

[14] Ueber die klinischen Verlauf und die anatomischen Veränderungen bei progressiver perniciöser Anämie mit Spinalstorungen, Neurol. Centralbl., 1902, xxvi, 620.

[15] Beiträge zur Pathologie der Leukämie, Ztschr. f. klin. Med., 1895, xx, 181.

[16] Sectionsbefund eines Falles von Morbus Ménièrei (Leukämie), Wien. med. Woch., 1896, xlvi, 919.

[17] Subacute Degeneration of the Spinal Cord, Brain, 1900, xxiii, 39.

[18] Rückenmarkserkrankung und Psychose bei perniciöser Anämie, Arch. f. Psychiatrie, 1909, xlv, 567.

[19] Nervous Symptoms and Morbid Changes in the Spinal Cord in Certain Cases of Profound Anemia, Medico-Chirurg. Trans., London, 1895, lxxxviii, 151.

reported cases dependent on chronic ergot poisoning, Tuczec,[20] Redlich[21]; carcinoma, Lubarsch,[22]; diabetes, Williamson,[23] Sandmeyer[24]; septicemia and ulcerative endocarditis, Nonne[25]; pellagra, Tuczec,[26] Marie,[27] Belmondo[28]; lathyrism; nephritis; malaria, Mayer[29]; shock; lead-poisoning, v. Monakow,[30] Oppenheim;[31] arsenic poisoning, Dana;[32] phosphorus poisoning; Addison's disease, Fleiner,[33] Brauer,[34] Kahlden;[35] tuberculosis, Summa;[36] syphilis; diphtheria, Bicheles,[37] Priesz;[38] typhoid fever and influenza, Redlich; scarlet fever, Billings;[39] chronic jaundice, Minnich;[40] chronic alcoholism, Braun,[41] Nonne,[42] Vierordt,[43] Redlich; leukemia, Kast, Müller,[44] Alt, Nonne,[45] Bloch and Hirschfeld;[46] bothriocephalus

[20] Ueber die Veränderungen im Centralnervensystem, special in den Hintersträngе bei Ergotismus, Arch. f. Psychiat. 1882, xiii, 99.

[21] Ueber einige toxische Erkrankungen der Hinterstränge des Rückenmarks (Ergotismus, Pellagra, Infektionskrankheiten, chronischen intoxicationen und bei den Polyneuritis, perniciösen Anämie, Leukämie, Diabetes, Addison'sche Krankheit, etc.), Centr. f. allg. Path., 1896, vii, 985.

[22] Hyperplasie und Geschwülste (Bedeutung des Carcinoms für den Gesamtorganismus), Lubarsch-Oestertag's Ergebnisse, etc., 1895, ii, 519.

[23] Changes in the Posterior Columns of the Spinal Cord in Diabetes Mellitus, Brit. Med. Jour., 1894, i, 398.

[24] Beitrag zur pathologischen Anatomie des Diabetes Mellitus, Deut. Arch. f. klin. Med., 1892, 381.

[25] Rückenmarksuntersuchungen in Fallen von perniciösen Anämie, von Sepsis, und von Senium, etc., Deut. Ztschr. f. Nervenheilk, 1898-99, xiv, 192.

[26] Klinische und anatomische Studien über die Pellagra, Berlin, 1883.

[27] De l'origine exogène ou endogène des lèsions du cordon postérieur étudiées comparativement dans le tabes et dans le pellagra, Sem. méd., 1894, xiv, 17, 28.

[28] Le alterazioni anatomishe del midollo spinalle nella pellagra, Riforma med., Napoli, 1889, v, 1532.

[29] Oppenheim's Diseases of the Nervous System, p. 159.

[30] Zur pathologischen Anatomie der Bleilähmung, Arch. f. Psych., 1880, x, 495.

[31] Zur path. Anat. der Bleilähmung, Arch f. Psych., 1885, xvi, 476.

[32] On Pseudotabes from Arsenical Poisoning, with a Consideration of the Pathology of Arsenical Paraylsis, Brain, 1886, v, 456.

[33] Ueber die Veränderungen des sympathetischen und cerebrospinallen Nervensystems bei zwei Falle Addison'sche Krankheit, Deut. Ztschr. f. Nervenheilk., 1891-92, ii, 265.

[34] Beitrag zur Lehre von den anatomischen Veränderungen des Nervensystems bei Morbus Addisonii, Deut. Ztschr. f. Nervenheilk., 1895, vii.

[35] Ueber Addison'sche Krankheit, Ziegler's Beiträge, 1891, x, 494.

[36] Ueber degenerative Veränderungen in Rückenmarke bei chronischer Lungenschwindsucht, Inaug. Diss., Freiburg, 1891.

[37] Post-diphtheritische Lähmung (Degeneration in die Rückenmarksträngen und hinter Wurseln), Arb. a. d. Inst. Obersteiner Wien, 1894.

[38] Beitrag zur Anatomie der Diphtheritischen Lähmung, Ztschr. f. Nervenheilk., 1894, vi, 95.

[39] The Changes in the Spinal Cord and Medulla in Pernicious Anemia, Shattuck Lecture, Boston, 1902.

[40] Zur Kenntniss der im Verlaufe der perniciösen Anämie beobachteten Spinalerkrankungen, Ztschr. f. klin. Med., 1892, xxi, 25, 246.

[41] Ueber die experimentelle durch chronische Alcoholintoxication hervorgerufenen Veränderungen im centralen und peripheren Nervensystem, Inaug. Diss., Tübingen, 1899.

[42] Klinische und anatomische Untersuchung eines Falles von Pseudotabes alcoholica, Jahrg. d. Hamb. Staatskrankenanst, 1892, ii, 91; Anatomische Befunde im Rückenmark bei Alkoholismus Chronicus Gravis, Deut. med. Woch., 1907, xxxiii, 166.

[43] Degeneration der Goll'schen Stränge bei einem Potator, Arch. f. Psych., 1886, xvii, 365.

[44] Ueber Veränderungen des Nervensystems bei Leukämie, Inaug. Diss., Berlin, 1895.

[45] Ueber Degenerationsherde in der weissen Substans des Rückenmarks bei Leukämie, Deut. Ztschr. f. Nervenheilk., 1896-97, x, 165.

[46] Zur Kenntniss der Veränderungen an Centralnervensystem bei der Leukämie, Ztschr. f. klin. Med., 1900, xxxix, 32.

anemia, Lichtheim; tetanus, Bonome,[47] Orr and Rows;[48] after extirpation of the adrenals experimentally, Tizzoni;[49] leprosy, Looft;[50] acute insanity; etc. Such a list offers a strong evidence in support of a toxic factor as the primary agent in producing the tissue changes. Old age has also been ascribed as a cause of similar spinal cord degeneration (Sanders,[51] Naka,[52] and others), changes having been noted particularly in the posterior columns. Leyden[53] discussed this problem of cord involvement in old age, and concluded that it was a process secondary to some ganglion cell atrophy, rather than a primary white matter degeneration. Ribail[54] traced the condition found in certain old subjects to a thickening of the membranes with the resulting local sclerosis, and Démange[55] concluded that the spinal cord lesions in old age results from a thickening around and of the walls of the vessels which, however, causes appearances differing widely from any system of sclerosis. Nonne, in ten cases of senile marasmus, found a glial thickening and nerve fiber atrophy in the posterior columns, and to a slight extent although less definite, in the lateral tracts. He also observed a non-inflammatory thickening of the vessels limiting their lumen. It might also be mentioned, although our observations are not sufficiently numerous to be conclusive, that of the cord lesions from four extremely old and very atrophied subjects, who presented at autopsy no other lesions than extreme general atrophy and some little arteriosclerosis, none showed areas of degeneration anywhere in the gray matter of the cord. The vessels occasionally did show a slight hyaline thickening but were not surrounded by any nerve tissue degeneration or glial proliferation. The nerve fibrils as a whole, seemed somewhat atrophied, but the cords were all remarkably and relatively well preserved. The part played by defective nutrition, *per se*, in the production of degeneration in the cord is doubtful. Oppenheim denies it, and Pugibet[56] did not find any marked change in the cords of subjects dead from chronic dysentery. Experimentally, the results of attempts to produce similar degeneration in the cord, have not been uniform. Grünfeld[57] failed to produce any result on

[47] Sulla alterazione del midollo spinale nel tetano, Arch. per le sc. med., Torino, 1891, xv, 15

[48] A Clinical and Experimental Investigation into the Lymphogenous Origin of Toxic Infection of the Central Nervous System, Brit. Med. Jour., 1907, i, 987.

[49] Ueber die Wirkung des Extirpation des Nebennieren auf Kanninchen, Ziegler's Beiträge, 1889, vi, 3.

[50] Beitrag zur pathologischen Anatomie des Lepra anesthet. insbesondere des Rückenmarks, Virchow's Archiv, 1892, cxxviii, 215.

[51] Alters Veränderungen in Rücknemark, Ztschr. f. Nervenheilk, 1900, xvii, 369.

[52] Die pathologische Anatomie des senilen Rückenmarks, Arch. f. Psych., 1906-07, xlii, 604.

[53] Die senilen Processe im Rückenmarkkrankheiten, 875, ii, 42.

[54] Observations pour servir à l'histoire de l'arachnitis et de la lepto-méningite spinale chronique—sclérose médullaire secondaire, Gaz. méd. de Paris, 1885, ii, 25, 57.

[55] Contribution à l'étude des scléroses médullaires d'origine vasculaire, Rev. de méd., 1884, 753.

[56] Des paralysies dans la dysentèrie et la diarrhée chroniques dans les pays chauds, Rev. de méd., 1888, viii, 110.

[57] Zur Frage ueber die Wirkung des Mütterkorns und seiner Bestandtheile auf das Rückenmarks der Thiere, Arch. f. Psych., 1890, xxi, 618.

the cord by means of ergot injections. Brauer, however, obtained definite lesions in the cord as the result of alcohol administration. Von Voss[58] produced severe anemia in animals, but was unsuccessful in producing any definite cord lesions. It must then be held that any toxic process, if sufficiently severe and prolonged, may cause a degeneration in the posterior tracts or even a development of a widespread degeneration. Gordon[59] and others have made the distinction, although not widely credited, that the hemolyzed substances set free by the action of a toxin of the blood are responsible for the changes in the cord.

Clinically, from the records available, it seems impossible to associate the extent of the cord lesion with the degree of anemia. It seems, however, reasonable to infer than when the tissues are fed by the altered blood of these anemic states, a condition analogous to ischemia exists and at such times changes in the nervous system would be likely to begin or extend. It would seem, therefore, that when the blood conditions are at their severest the most critical neurological examination should be made and repeated at frequent intervals.

In the evolution of the literature of the spinal cord changes in pernicious anemia, several different pathological processes have been described as being the essential etiological factor. Thus, coalition of multiple small areas of hemorrhage to form a tract sclerosis was advocated by many of the earlier writers, Teichmüller,[60] Minnich, van Noorden,[61] Petrén,[62] etc. Myelitis was the essential process according to Nonne.[63] Boedecker and Juliusberger,[64] Marie, Rothman,[65] etc., described it as endogenous, being the sequence of an involvement of the ganglion nerve cells. Thickening of the vessel walls with resulting defective nutrition of the cord was considered by Nonne, Minnich, Redlich,[66] and others. It has also been suggested by Schmaus,[67] and others that the immediate destruc-

[58] Anatomische und experimentelle Untersuchungen über die Rückenmarks-Veränderungen bei Anämie, Deut. Arch. f. klin. Med., 1897, lxviii, 489.

[59] Histological Changes in the Spinal Cord in Pernicious Anemia, New York Med. Jour., 1909, xc, 80.

[60] Ein Beitrag zur Kenntniss der im Verlaufe der perniciösen Anämie beobachteten Spinalerkrankung, Deut. Ztschr. f. Nervenheilk., 1896, vii, 385.

[61] Untersuchungen über schwere Anämien, Charité Annalen, 1891-92.

[62] Bidrag till Kännedomen om ryggmärgsförändringar vid perniciös anemi, Nord. Med. Ark., Stockholm, 1896, ix, No. 7, 1.

[63] Perniciösen Anämie beobachteten Spinalerkrankungen, Arch. f. Psych., 1893, xxv, 421; Weitere Beiträge zur Kenntniss der im Verlaufe der perniciösen Anämie beobachteten Spinalerkrankungen, Deut. Ztschr. f. Nervenheilk., 1894-95, vi, 313.

[64] Casuistisches Beitrag zur Kenntniss der anatomische Befunde bei spinaler Erkrankung mit progressiven Anämie, Arch. f. Psych., 1898, xxx, 372.

[65] Die primären combiniter Strängerkrankungen des Rückenmarks, Deut. Ztschr. f. Nervenheilk., 1895, vii, 171.

[66] Ueber eine eigenthümliche durch Gefässdegenerationen hervorgerufene Erkrankung der Rückenmarks Hinterstränge, Ztschr. f. Heilk., Berlin, 1891, xxi, 247.

[67] Zur pathologischen Anatomie der Seitensträngerkrankung bei Tabes Dorsalis, Deut. Arch. f. klin. Med., Leipsig, 1889-90, xliv, 113.

tion of the medullary sheaths and degeneration of the neuraxons is dependent on lymph stasis due to impoverished condition of the blood and its excess in lymph elements. All these various conditions could not, for obvious reasons, be responsible for the appearance found in the cord in pernicious anemia, nor could any evidences of such processes be observed in either of our two cases. Excessive fatigue is also a very doubtful factor, as it has been shown that nerve fibrils are practically inexhaustible and certainly less so than the ganglion cells or muscles. The process, agreeing with the conclusions of most modern authors, appears to be that of a degeneration with more or less replacement gliosis, and this accounts best for its irregular, imperfectly systemic, and incomplete character.

To account for the degeneration occurring especially in the posterior columns, Orr and Rows have shown that in tabetic conditions the degeneration is particularly marked at the point of entrance of the nerve roots. Also by injections of tetanus toxin and various bacterial emulsions into nerves they have demonstrated how the toxic agents ascend to the cord. They consider that the immunity of the nerves is due to the enveloping neurilemma and that only in the unprotected portion of the system where the nerve fibrils are contained in the cord, can the toxins produce any effect. It is certainly, however, somewhat difficult to apply this to either of our cases; moreover, in both of our cases the posterior roots were not altered. It seems more natural to assume a greater vulnerability to degenerative processes in certain areas of the spinal cord than in others.

In connection with these cases it has to be, to some extent, considered, just how far postmortem conditions can simulate any system lesion. These postmortem processes, however, do not produce the peculiar degenerative vacuolization nor the hyaline appearances of gliosis. In this connection the study of a series of such postmortem conditions in the cord at various intervals after removal from the body, was undertaken, with the result that system changes or appearances such as have been described were not observed.

In the consideration of the cavity formation in the lower dorsal region in Case I, there perhaps lies the one point of interest which permits of these cases being put on record. The cavity, as has been noted, was situated on the left side of the cord, between the tenth dorsal and first lumbar segments, and occupied almost exclusively the gray matter, particularly of the anterior horn. This destruction and cavity formation corresponded almost entirely to a branch of the anterior spinal artery, This vessel supplies the greater part of the gray matter, while most of the rest of the cord is nourished by the pial vessels. It was apparently a thrombosis in one of the small veins which accounted for all the œdematous separation of the tissues and the comparative absence except where the cavity was

largest, of any necrosis of tissue. This process probably was produced in the last few hours of life. It was difficult to be exact as to the duration of the process, as no clinical symptoms had been evident. The absence of clinical symptoms is explained by the location of the lesion and by the fact that the patient was comatose for some time before death.

Several examples of cases of thrombosis of the spinal cord vessels have been mentioned by various authors (Dana, Petrén[68]), and Hawthorne[69] has advocated that many of the cerebral and ocular complications of pernicious anemia (retrobulbar neuritis, ocular paralysis, optic neuritis, etc.) are due to vascular thrombosis. The appearances, however, described by these authors, have not been quite similar to what was found in our cases. These have been, in the majority of cases, of the nature of a hemorrhagic necrosis of the gray matter, particularly of the anterior horn. Experimentally, such conditions have also been produced by aseptic emboli, by Lamy;[70] also Ehrlich and Brieger,[71] Münzer and Wiener,[72] Singer and Münzer,[73] and Spronck[74] have produced similar changes by clamping the aorta in rabbits just behind the renal artery. Singer and Münzer showed in such conditions that areas of the posterior columns may become degenerated, but in our case the degeneration in the posterior tracts, besides being bilateral, was evidently of much older date than the cavity in the gray matter in the lower dorsal region.

Bulloch[75] (1892) described, in a case of pernicious anemia, hyaline masses in the cord chiefly involving the anterior horns of the gray matter, and referred this to an exudation from the adjacent vessels. Henenberg[76] (1899) described two cases of combined sclerosis in which there were found cavities in the spinal cord. In one case the cavity was central and in the other it occupied the centre of one of the anterior horns. He considered thrombosis as the essential factor in their production.

The cavities in the spinal cord in syringomyelia, and the simple

[68] Fall von akuter Infektionskrankheit mit Thrombose in den pialen Gefässen, etc., Nord. Med. Ark., 1898, ix, No. 7, 1.

[69] An Address on the Cerebral and Ocular Complications of Anemia and the Probable Relationship of These to Thrombosis, Lancet, 1908, ii, 857.

[70] Lésions médullaires expérimentales produites par les embolies aseptiques, Arch. de physiol. norm. et pathol., 1897, ix, 184.

[71] Ueber die Ausschaltung des Lendenmarksgrau, Ztschr. f. klin. Med., 1883, vii, Supp. Heft, 155.

[72] Beiträge zur Anatomie und Physiologie des Centralnervensystems, Arch. f. exp. Path. u. Pharmak., 1895, xxxv, 113.

[73] Beiträge zur Anatomie des Centralnervensystem, Akad. d. Wissenschaften, Wien, Denkschriften, 1890, lvii.

[74] Contribution à l'étude expérimentale des lésions de la moelle épinière déterminées par l'anémie passagère de cet organe, Arch. de phys. norm. et path., 1888, 389.

[75] Hyaline Degeneration in the Spinal Cord, Brain, 1892, xv, 411.

[76] Beitrag zur Kenntniss der combinite Strängdegeneration, sowie der Höhlenbildung im Rückenmark, Arch. f. Psych., 1889, xxxii, 550.

serous cysts which from time to time have been described in the cord and brain, may, some of them at least, be possibly of similar origin. Again, certain cases of pernicious anemia have been described as terminating with fulminating and intense nervous symptoms, and in these the occurrence of multiple thrombi and resulting extensive cord destruction is very probable.

A hemorrhage into the spinal cord, such as might easily occur in pernicious anemia, might, of course, produce localized paralytic symptoms, as a case reported by Riggs,[77] but it would be likely to have the definite location and completeness of destruction of an embolism or thrombosis.

Had Case I, of the two here reported, survived, the final result would probably have been exactly like acute anterior poliomyelitis. It seems, moreover, peculiar that such conditions do not more frequently occur, particularly in various states of defective vitality or in the course of some general infective process.

[77] Paraplegia Arising from Hemorrhage into the Spinal Canal, due to Pernicious Anemia, Jour. Nerv. and Ment. Dis., 1896, xxi, 554.

REVIEWS.

MODERN MEDICINE. ITS THEORY AND PRACTICE. IN ORIGINAL CONTRIBUTIONS BY AMERICAN AND FOREIGN AUTHORS. Edited by WILLIAM OSLER, M.D., Regius Professor of Medicine in Oxford University, England; Assisted by THOMAS MCCRAE, M.D., Associate Professor of Medicine and Clinical Therapeutics in the Johns Hopkins University, Baltimore. Vol. VII; pp. 969; 37 plates and 12 text illustrations. Philadelphia and New York: Lea & Febiger, 1910.

THE seventh and concluding volume of Osler's *Modern Medicine* is devoted to a consideration of diseases of the nervous system. It includes a wide range of topics, all of which are ably handled by well-qualified authors, as follows: An introduction, by Lewellys F. Barker; diseases of the motor tracts, by William G. Spiller; combined system diseases, by Colin K. Russell; sclerosis of the brain and diseases of the meninges, by Edwin Bramwell; diffuse and focal diseases of the spinal cord, by E. Farquhar Buzzard; topical diagnosis of diseases of the brain and aphasia, by Joseph Collins; diseases of the cerebral bloodvessels, by Henry M. Thomas; tumors of the brain and meninges and hydrocephalus, by Harvey Cushing; diseases of the peripheral nerves, by Gordon M. Holmes; diseases of the cerebral nerves, by E. W. Taylor; paralysis agitans, chorea, choreiform affections, and infantile convulsions, by D. J. McCarthy; acute encephalitis and brain abscess, by E. E. Southard; syphilitic and parasyphilitic diseases of the central nervous system, and amaurotic family idiocy, by Bernard Sachs; neurasthenia, and the traumatic neuroses and psychoses, by Charles W. Burr; and migraine, neuralgia, professional spasms, occupation neuroses, tetany, and hysteria, by Smith Ely Jelliffe.

Critical comment of all these subjects is altogether out of the question within the available space; but fully conscious of the excellence of a number of articles not again specifically mentioned, one may perhaps direct special attention to several of much importance. Thus, for instance, Barker, in his introductory chapter, discusses in an illuminating manner the senses and their symptomatology; disturbances of sensation; anomalies of sense perception; disturbances of complex psychic processes, of motility, and of coördination; and anomalies of speech and writing, of reflexes, and

of vasomotor, secretory, and trophic functions. Altogether the chapter is an example of careful writing and clear reasoning, and it is replete with information of a diversified but related nature, the details of which are here brought together and rendered available.

Spiller's chapter on diseases of the motor tracts comprises a lucid and accurate account of progressive spinal muscular atrophy, progressive spinal muscular atrophy of childhood, amyotrophic lateral sclerosis, lateral sclerosis, hereditary spastic spinal paralysis, unilateral ascending and unilateral descending paralysis, progressive neural muscular atrophy, interstitial hypertrophic neuritis of childhood, progressive bulbar palsy, and muscular dystrophy. All of the discussions bear the imprint of Spiller's well-known care and painstaking, his thorough knowledge of the subject, and his efforts to explain clinical phenomena in terms of anatomical lesions. For instance, he discusses amyotrophic lateral sclerosis as apart from progressive spinal muscular atrophy and from chronic or subacute poliomyelitis. Bramwell has enhanced the value of his chapter on diseases of the meninges by a short but excellent section on lumbar puncture and the examination of the cerebrospinal fluid. Buzzard, in his chapter on diffuse and focal diseases of the spinal cord, discusses the localization of non-systemic diseases of the cord and the cauda equina, and illustrates the practical application of the information thus imparted in the diagnosis of compression and tumors of the spinal cord. His discussion of the symptomatology and diagnosis of tumors of the cord well repays careful reading, and should lead to an early diagnosis of these growths—at a time when many of them are amenable to surgical procedures. Of Collins' chapter on the topical diagnosis of diseases of the brain, one can well say it is worth the reading; the information therein contained is essential to an accurate diagnosis of irritative and destructive lesions, and it is so presented as to be readily comprehended by those less well informed than its author. This may also be said of Collins' chapter on aphasia, wherein one finds a thorough and critical discussion of the current opinions on the intricate subject of aphasia, as well as of the revision of the subject proposed by Marie.

Thomas, among other subjects, discusses the important condition of apoplexy. He uses the term in what he describes as its original symptomatic sense—"to strike off or be disabled by a stroke;" and he includes therein cerebral hemorrhage, thrombosis, and embolism. The article, which comprises seventy-one pages, is one of the most valuable in the volume, and is embellished by a number of statistics, especially those relating to 740 patients studied at the Johns Hopkins Hospital. Harvey Cushing's chapter on tumors of the brain and meninges is especially important and valuable, since it has been contributed by one who has achieved an international reputation in cerebral surgery and is a record of personal observation and study of 136 cases.

As syphilitic and parasyphilitic diseases of the nervous system, Sachs includes cerebrospinal syphilis, acute and chronic specific myelitis, syphilitic spinal paralysis (Erb's type), tabes dorsalis, and general paresis. There can be but little adverse comment in including tabes dorsalis as a syphilitic disease, although writers now and then report cases in which syphilis can or seems to be excluded. Sachs himself has seen only one case in which he was certain of the absence of syphilis, and since the use of the Wassermann reaction the demonstration of syphilitic infection in cases that otherwise would have passed as non-syphilitic is becoming common. Burr's contribution on neurasthenia and the traumatic neuroses and psychoses needs to be read to be appreciated; no one who reads the chapter will for a moment doubt what the author means, and, what is perhaps the greater compliment, he will almost always agree with him.

Smith Ely Jelliffe contributes fifty-seven very interesting pages on hysteria. Herein one will find at least mention of the many and varied opinions and divers writings that have illuminated (and clouded) the subject from pre-Christian to the present times—the views of the Charcot school, the studies of Pierre Janet, Freud's hypotheses and accentuation of the sexual basis of the hysterical phenomena, Babinski's ideas especially concerning the suggestibility of the manifestations, and much more, including some illuminating ideas and comments by the author himself. It is interesting to some of us to read: "In practice it may take months or years to fully analyze some hysterical cases. When fully analyzed, the patients become cured—the analysis has been a catharsis." The discussion of the treatment of hysteria is excellent, and should be read by general practitioners.

The work is done; three years have elapsed between the publication of the first and the seventh volumes—a record of which the editors and the publishers may well be proud. Commenting upon the first volume, we said that it was well conceived, excellently planned, and splendidly executed; we have entertained a similar opinion as each succeeding volume has come to hand, and now that the work is finished we are finally confirmed in the opinion. The many contributions that go to make up the seven volumes attain a high average level; there are very few, if any, that are mediocre; many are of unusual excellence and in the broadest sense authoritative. The work as a whole is a repository of our knowledge of the present state of medicine; the plan of the editor who designed the work primarily for the practitioner who wishes to keep himself informed of the existing state of our knowledge in clinical medicine has been fulfilled; so that we may say of the entire work, as we did of the first volume, it may be warmly recommended to the physician as replete with the practical applications of science to medicine, mirroring the clinical manifestations of disease, and helpful and resourceful in suggestions for treatment. A. K.

THE MALARIAL FEVERS, HEMOGLOBINURIC FEVER, AND THE BLOOD PROTOZOA OF MAN. By CHARLES F. CRAIG, M.D., Captain, Medical Corps, U. S. Army. Pp. 477; 4 colored plates, 25 clinical charts, and 28 photomicrographs and drawings. New York: William Wood & Company, 1909.

THE discovery of the true cause of malaria by Laveran gave renewed impetus to the study of this infection. As a result, there has grown up during the past thirty years an extensive literature upon all phases of malaria. However important much of this literature may be, a large part of it is of necessity inaccessible to the majority of practitioners, especially to most English speaking ones. Yet, as our invasion of the tropics goes steadily forward year by year, the ever-increasing importance of the malarial infections makes it essential that the facts discovered by so many able workers in this field should be squarely placed before the entire medical profession. To review critically the work of others and to set forth clearly our modern conception of malaria is an undertaking only to be assumed by one conversant with the literature and at the same time familiar with the clinical aspect of the subject through wide personal experience. It is fortunate, therefore, that this difficult task has been undertaken by so painstaking a student of malaria as Dr. Craig, whose experience in dealing with over 5000 cases of malaria of all forms in various parts of the world has rendered him competent to write with authority upon the malarial fevers.

Craig divides the malarial plasmodia into four distinct species: (1) Plasmodium malariæ (quartan parasite); (2) Plasmodium vivax (tertian parasite); (3) Plasmodium falciparum (tertian estivo-autumnal parasite); and (4) Plasmodium falciparum quotidianum (quotidian estivo-autumnal parasite). In view of the controversy and confusion which exists as to the classification of the malarial parasite, it is interesting to note that Craig unhesitatingly states that there are two distinct species of estivo-autumnal plasmodia, the tertian and quotidian.

He devotes the first part of the book to the general question of etiology, but more particularly to a consideration of the morphology and development of the plasmodia within man and the mosquito. The detail with which he enters into the differentiating characteristics of the various species of malarial organisms when found in human blood should make it possible for even the inexperienced student to recognize the different forms of infection. Unlike some observers, he holds that the sporogenic forms of the plasmodia, the gametes, are not introduced as such by the infected mosquito, but arise within the human blood after the asexual forms have sporulated for some time. He refutes and dismisses as worthless all evidence which attempts to show that malaria is transmitted

to man in any way save by the bite of infected mosquitoes. Further, he denies the existence of congenital malaria, and states that infection of the fœtal portion of the placenta by malarial parasites does not occur.

After devoting several chapters to a careful review of the pathology of malaria, he enters upon the discussion of the clinical aspects of the subject. He opposes the usual clinical classification of the malarial fevers into intermittent, remittent, and continuous, on the ground that any variety of malarial infection may exhibit any one of these three types of fever. He prefers to divide malaria clinically according to the variety of the infecting parasite, thereby doing away with much confusion by bringing into accord the clinical and etiological classifications of malaria.

In speaking of pernicious malaria Craig follows the usual custom of designating the various forms according to the symptoms which preponderate. He makes it clear, however, that pernicious malaria is not a separate disease due to any particular species of plasmodium. He emphasizes the fact that any of the forms of malarial parasites which are capable of causing the mildest types of infection, may, under favorable conditions, give rise to the rapidly fatal pernicious forms of malaria. The frequency of latent malaria, a form of infection in which some type of the plasmodium is found in the blood of individuals who exhibit no symptoms of the disease, is shown by the author's experience. He found that out of 1297 cases of malaria in Americans, 307, or nearly 24 per cent., were latent infections. The importance of recognizing such cases from the standpoint of prophylaxis becomes at once apparent.

It is well known that primary malarial infections are prone to recur. How these recurrences are brought about, in the absence of any reinfection, has occasioned considerable controversy. As a result of careful study, Craig has made some interesting observations upon this point. He has been led to conclude that within the blood of man malarial infections are perpetuated and hence tend to recur, through a process which he terms intracorpuscular conjugation. Briefly, this consists in "the complete and permanent union of two unpigmented plasmodia within the red blood corpuscles." The process is similar to conjugation as observed at some period in the life cycle of nearly all protozoa, the purpose of which is the restoration of the property of reproduction in an exhausted organism.

The chapters dealing with prophylaxis and treatment, although carefully prepared and well written, contain little that is not already well known. The personal observations of the author upon the changes induced by quinine in the different species of malarial plasmodia are noteworthy. Throughout the discussion of treatment his dictum is that success in treating malaria depends more upon the manner in which quinine is given than upon the quantity of the drug administered. He strongly advocates the prolonged

administration of quinine in divided doses rather than the method of giving a single massive dose before the expected paroxysm.

The closing section of the book is devoted to a consideration of the blood protozoa in man other than the plasmodium of Laveran, and of hemoglobinuric fever. In regard to the latter disease the author is emphatically opposed to the theory that it is due either to malaria or to quinine. He looks upon hemoglobinuric fever as "a disease *sui generis*, caused by a hitherto undiscovered organism, probably of protozoal nature."

Throughout the book one is impressed by the fact that the author's large experience with the disease has enabled him to form an individual opinion upon nearly all questions of importance. Whenever his observations are at variance with those of other workers he has the courage uncompromisingly to state his position. The work contains only the typographical errors to be expected in a first edition. Some of those noted occur on pages 93, 102, 218, 223, and 231. The value of the book as a work of reference is greatly increased by the bibliographies appended to each chapter. There is no question that an enormous amount of painstaking labor has been expended in gathering the material which forms the basis for most of the author's statements. Dr. Craig has furnished the profession with an extremely useful and at the same time interesting book upon a subject of increasing importance. If his efforts meet with the widespread recognition which is his due, it is safe to predict that many of the vague uncertain conceptions of the malarial fevers still held by some persons will be radically altered, much to the benefit of many communities. G. M. P.

OPERATIONS UPON THE UTERUS, PERINEUM, AND ROUND LIGAMENTS. By W. J. STEWART MCKAY, M.B., M.CH., B.SC. Senior Surgeon, Lewisham Hospital for Women and Children. Pp. 454; 148 illustrations. New York: William Wood & Co., 1909.

THIS is a well-printed and most profusely and handsomely illustrated book. The title is, however, somewhat misleading, since the operations upon the uterus consist merely of curettage, of those procedures aiming to correct a retrodisplacement, and the operative treatment of dysmenorrhœa. The volume opens with a lengthy chapter upon the anatomy of the vulvoperineal region, together with a discussion of the choice of operative procedures for the repair of the injuries of this region. The author believes in the H-shaped operation of Tait for the repair of complete tears and in Emmet's method for lesions of the vaginal sulci, unless there be marked rectocele, when the Hegar is advised. In discussing the

repair of complete rectal tears, he says that "this accident is very serious and must be attended to at once, otherwise the patient will have no control of her motions." In parentheses we may be forgiven for saying that we object to the last word in the passage quoted, believing that there are several better ones in the English language. A more important criticism, however, relates to the alleged necessity for immediate repair; in a rather extensive experience we have learned that immediate operation is not a necessity, and we are entirely convinced that if it is deemed inadvisable to repair without ether, it is better to wait for several days, since there is no question that to give an anesthetic immediately after placental delivery is dangerous practice. Moreover, a tired woman, tired doctor, poor light, and inefficient assistants do not tend to the production of good surgery. We have been greatly interested and surprised to note that the author advises that ether be given to remove vaginal stitches. We cannot imagine any possible reason for advocating this as a routine; we have never been obliged to resort to anesthetics for the removal of either the cervical or vaginal stitches, and we consider that it is unjustifiable.

In the description of the operative relief of cystocele we note a mistake due to oversight, the author stating, upon the authority of Hirst, that the deep transversus perinei is the strongest support of the anterior vaginal wall. The muscle of the urogenital trigonum, described by Waldeyer, and upon the importance of which Hirst always lays great stress, is of course the muscle meant by the author. We are sorry that we are unable to refer to this section with any degree of satisfaction. The description is meagre and the operative treatment advised is antiquated and inadequate. In his consideration of the repair of cervical injuries, a satisfactory exposition, we are again met with the astounding statement that "to remove them [that is, cervical sutures] an anesthetic should always be administered." The immediate repair of cervical lacerations is, we are glad to see, strongly advised against, as is also any operative procedure for the repair of old perineal lacerations during the lying-in period.

While not agreeing with certain minor points in his operative technique, we take pleasure in commending the chapter, of over one hundred pages, upon displacements of the uterus. The succeeding chapter upon uterine curettage is, in general, satisfactory, though we do not fancy the delay in emptying the uterus, advised by the author, in cases of inevitable or incomplete abortion, as we feel that repeated tamponade predisposes to sepsis. The next chapter is devoted to a consideration of curettage of the uterus; over one hundred pages are devoted to this subject, none too many, and it is a very satisfactory thesis, except for a footnote to page 396, in which the author states that in treating a case of streptococcic infection he transfused blood from the husband. Upon

the basis of this one case he draws the conclusion "that fresh blood transfused will stop the rigors and reduce the temperature of puerperal septicemia." Aside from the fallacy of forming his judgment upon entirely insufficient data, we feel that the author, in the light of recent investigations, has made an unfortunate statement.

The volume closes with a chapter comprising two pages and four illustrations upon dysmenorrhœa. The author advocates the splitting of the posterior cervical lip as the best treatment for obstructive dysmenorrhœa, the only variety categorically mentioned except that due to posterior displacements of the uterus. We cannot honestly say that we think that this book is necessary to the advancement of medicine or that it is the best exposition of the limited portion of gynecology of which it treats. W. R. N.

SURGICAL DIAGNOSIS. By ALEXANDER BRYAN JOHNSON, Ph.B., M.D., Professor of Clinical Surgery in the Columbia University Medical College, New York. Three volumes (pp. 810, 777, 810); 5 colored plates, and 784 illustrations in the text. New York and London: D. Appleton & Co., 1909.

THIS ambitious work on *Surgical Diagnosis* is dedicated "To Doctor Charles McBurney, the greatest surgical diagnostician the author has ever known;" it "embodies to some extent the impressions gained during an experience extending over a period of nearly twenty-five years;" and in preparing it "the author has had constantly in mind the needs of the *practitioner of general medicine*, and it is to him especially, he believes, that this work will be found valuable."

The subjects in Vol. I are treated in the following disorder: Wounds, infections, joints, bones, soft parts, tumors, fractures and dislocations, syphilis, leprosy, bloodvessels, *x*-rays, head, brain, ear, mouth (no eye), neck, œsophagus, thorax, breast, abdomen, and peritoneum; Vol. II, stomach, intestines, appendix, liver and biliary passages, spleen, pancreas, rectum, hernia, kidney, ureter, bladder, and genitals; Vol. III, spine and spinal cord, nerves, pelvis, shoulder, arm, elbow, forearm, wrist and hand, hip, thigh, knee, leg, foot and ankle. In an Appendix are included various matters, among which may be mentioned additional notes on polycystic kidney, rabies, parathyroid glands, Spirochæta pallida, acute rheumatic fever, exophthalmic goitre, operative indications in benign diseases of the stomach, lesions of the brain, gunshot wounds, serum diagnosis of syphilis, and status lymphaticus—in the remarkable succession named.

The volumes are exceedingly inclusive and tediously diffuse;

minute directions for blood examinations are given, and bacteriological methods and gastric and urinary analyses of extreme complexity are described in their utmost details. Much of this matter, we believe, should have been omitted; the tests cannot be made by the general practitioner himself, but only by trained pathologists, and these will not look for aid to a work on surgical diagnosis professedly written for the use of the former. Moreover, all these methods are quoted, often *verbatim*, from other books or recent journal articles.

The author is at his best when dealing with purely clinical diagnosis, in which, from his large experience, he has become an adept, and a worthy pupil of his skilled master. An effort has been made to include enough pathological data to be of service in making a true diagnosis; for the author seems to recognize that without a knowledge of the underlying pathological processes diagnosis is frequently a matter of happy (or unhappy) guesswork. In the first and second volumes he keeps to his subject of diagnosis with fair constancy; but in the third volume there is evidenced a tendency to discourse on therapeusis, which, so far as we can see, serves no other purpose than to fill an unnecessary number of pages; thus, an entire page is devoted to the *operative treatment* of fracture of the patella, another entire page to the treatment of fractures of the tibia, and two pages to the operative treatment of gastric lesions; and throughout the three volumes numerous case histories are quoted at length, which have much more interest from considerations of prognosis and treatment than from those of diagnosis.

The work gives the impression of having been written for the mere sake of writing a book, of making it as large as possible, and of using as many as possible of a large collection of illustrations assembled through many years by the author, his colleagues, and friends. The figures do not always appear to have been chosen with a view to elucidating the text, and in a number of instances the author has been unable in any way to bring the subject illustrated in the figures into his text, no reference to them, not even a remote reference, being discoverable. Yet many of the figures are very apposite illustrations of the text; and if only the author had not been swamped with his material, or if he had made illustrations to explain the text, instead of attempting unhappily to construct a book around the illustrations, the result would have been more satisfactory. There is a series of 32 figures of museum specimens (dried bone) of healed fractures of the humerus; yet the author has to apologize, in his text, for inability to use more than a "fraction" of the total number of specimens of this kind which were placed at his disposal. But there is no further reference in the text to this series of figures, which are of infinitesimal value for the purposes of diagnosis. Their places could well have been taken by five or six skiagrams of typical fractures before reduction.

The amount of space devoted to the various sections of the work is not always in relation to their importance. While the lesions of the liver, gall-bladder, pancreas, and spleen are discussed in less than 75 pages, no less than 216 pages (13 chapters) are devoted to the kidney and ureter, and 220 pages (14 chapters) to the bladder and genitalia. The female genitalia are not included.

Some of the chapters have a bibliography appended (for no apparent reason, as others have not), and each volume has at the end an alphabetical list of the authors cited, as well as an index; while the third volume has general indices to the whole work. It is interesting to see, in the general index of authors, that the writer of the volumes is cited as an authority no less than four hundred and sixty-four times, the mere list of references to the pages where he is quoted occupying nearly an entire page of the index; those to whom the volumes are addressed surely will emulate the rustics of whom Goldsmith wrote:

> "And still they gazed, and still the wonder grew
> That one small head could carry all he knew."

The work, we believe, could be condensed into two volumes, possibly into one large volume, with advantage; the laboratory tests could be omitted altogether; the case histories and the long *verbatim* quotations from recent journal articles might be given in abstract; and with efficient editing (which the volumes have not had) and rearrangement of the subject matter the work would assume a form much more apt to be received favorably by the profession.

A. P. C. A.

A Text-book of the Practice of Medicine. By James M. Anders, M.D., Ph.D., LL.D., Professor of Medicine and Clinical Medicine in the Medico-Chirurgical College, Philadelphia. Ninth edition; pp. 1326; 82 illustrations. Philadelphia and London: W. B. Saunders Company, 1909.

The new edition of Dr. Anders' *Practice of Medicine* represents a thorough revision of those that have gone before. Obsolete matter has been eliminated, and matter that means progress has been incorporated. A list of new or rewritten matter, were it printed, would be long; it comprises minor or major changes and additions to almost every page of the book. The section on tropical diseases has been enlarged. Having reached a ninth edition, the book, of course, is one of the standard volumes of the subject of which it treats. A. K.

PROGRESS

OF

MEDICAL SCIENCE.

MEDICINE.

UNDER THE CHARGE OF

WILLIAM OSLER, M.D.,

REGIUS PROFESSOR OF MEDICINE, OXFORD UNIVERSITY, ENGLAND,

AND

W. S. THAYER, M.D.,

PROFESSOR OF CLINICAL MEDICINE, JOHNS HOPKINS UNIVERSITY, BALTIMORE, MARYLAND.

The Cultivation of Tubercle Bacilli Directly from the Sputum by the Use of Antiformin.—The fact that the cultivation of tubercle bacilli directly from the sputum has never been an easy task makes any new method unusually interesting. BROWN and SMITH (*Jour. Med. Research*, Boston, 1910, xxii, 517) have made use of antiformin. The technique is very simple. Equal parts of 30 per cent. solution of antiformin in water and the sputum were thoroughly mixed in a sterile tube and then centrifugalized, the supernatant fluid decanted, and the sediment mixed with sterilized distilled water. This process was repeated three times. The sediment was then scattered over the surface of Dorset's egg medium and placed in the incubator. The results are summarized as follows: Of 50 specimens of sputum examined, 35 showed tubercle bacilli on microscopic examination. Positive cultures were contained in 33, or 97 per cent. of these 35 specimens, and in 4 of 15 specimens in which no tuberle bacilli were found microscopically. Guinea-pigs inoculated with these 37 cultures developed tuberculosis in every instance.

An Investigation of the Acid-fast Bacteria Found in Human Feces, with Especial Reference to their Presence in Tuberculosis.—In reviewing the literature on this subject, ALEXANDER (*Jour. Hygiene*, 1910, x, 37) finds it well established that acid-fast bacilli may be present in the feces in cases of pulmonary and intestinal tuberculosis, and that their presence, though in relatively large numbers, does not necessarily imply ulceration of the intestine. However, it has not been shown that these acid-fast bacilli are tubercle bacilli. The author injected subcutaneously into guinea-pigs, of the average weight, human feces in the average amount of 0.02 gram. He found that this was the largest amount that could with safety be injected. The feces of 23 cases of pulmonary

tuberculosis in which acid-fast bacilli had been demonstrated by the microscope were proved capable of infecting guinea-pigs with tuberculosis. An examination, microscopically, of 129 samples from non-tuberculous individuals revealed no acid-fast bacilli. From this we would infer that all acid-fact bacilli in the feces are tubercle bacilli. He emphasizes the fact that it is more difficult to demonstrate tubercle bacilli in constipated than in diarrhœal stools. In 74 cases the bacillus was demonstrated 52 times. These cases included bone, joint, meningeal, skin, glandular, pleural, pulmonary, and miliary tuberculosis.

An Experimental Study of the Blood Serum in Uremia.—CAWADIAS (*Comp. rend. Soc. de biol.*, 1910, lxviii, 976) has injected the blood serum of uremic patients into the peritoneal cavity of rabbits and produced immediately such phenomena as convulsions, paralyses, dyspnœa, hypothermia, even death. Normal serum will produce toxic symptoms, but much larger doses are necessary than of the serum of a uremic patient. Even the sera of uremic patients vary in toxicity, the serum of a uremic in convulsions being high in the scale. Cawadias emphasizes the fact that the serum of a well individual differs from that of a uremic only in degree of toxicity. The results of injection are the same if only the right amount is used. The conclusion is drawn that in uremia there is no new poison, but a preëxisting toxic substance whose action becomes more intense.

Fixation of the Complement in Hydatid Disease.—CHAUFFARD and VINCENT (*Bull. et mém. de la Soc. méd. d. hôp de Paris*, 1910, xxvii, 859) report a case of hydatid cyst of the liver with eosinophilia, in which the fixation of the complement failed. The history pointed to a duration of three years. The eosinophilia disappeared the evening of operation, whereas, eleven days later the reaction of the fixation of the complement was positive. Chauffard and Vincent conclude that there are cases of hydatid cysts (1) with all biological reactions positive, both eosinophilia and reaction of fixation. As a rule, the eosinophilia disappears after operation. On the other hand, cases (2) occur in which these reactions are negative before operation but appear after it. Finally, there exist (3) intermediary types with incomplete or dissociated reactions. They are of two forms—reaction of fixation positive without eosinophilia, and the opposite. After operation the positive reaction disappears and the negative becomes positive.

The Specific Treatment of Carcinoma.—COCA and GILMAN (*Philippine Jour. Science*, 1910, iv, 391) have used a vaccine in the specific treatment of carcinoma. This was prepared as follows: The tumor tissue, immediately after excision, was cut up with scissors and passed ten times through a vaccine grinder which had been sterilized by successive rinsing through pure carbolic acid, absolute alcohol, and sterile salt solution. After the first passage through the grinder an equal amount of sterile salt solution was added. By thorough grinding of the tissue, practically all the tumor cells were completely broken up, so that the decanted fluid contained all the protoplasm and nuclear substances of these cells. In order to get rid of the shreds of fibrous tissue the thick creamy fluid was centrifugalized at a high speed for three

minutes and decanted. The injection was made preferably within six hours after the addition of the carbolic acid, which was made in the strength of 1 c.c. of 5 per cent. carbolic acid to every 10 c.c. of fluid. This was an antiseptic measure. The largest amount injected at one time was about 20 to 25 grams. The degree of local reaction of the tissues to the injection has varied from absolutely none whatever in a case of Hodgkin's disease to abscess formation in two cases of carcinoma. One of these abscesses was sterile. In 3 cases affected with carcinoma, such injections have been followed by a softening and disappearance of tumor masses measuring in diameter from 2 to 4 cm. In the fourth case a rapidly growing lump, which had appeared in the site of an excised cancer two days after treatment, was shown a week later to be a firm encapsulated mass of dying epithelial-like cells. Three other surgically inoperable cases have remained from five weeks to six months free from recurrence of disease. Seven other cases have remained free from a return of the disease for from one to seven months. Two of these were designated as curable by operation, three as probably curable, and two as of doubtful curability. In only one case has disease returned after treatment. Here the material used for vaccine was rendered inert by too vigorous disinfection. Large injections of tumor material seem to cause a rapid disappearance of cachexia. The efficacy of the treatment is indirectly proportional to the amount of malignant tissue left in the body, and within certain limits directly proportional to the amount of tumor cell substance injected and to the number of injections made. Apparently carcinoma in one individual may be successfully treated with injections of material from a carcinoma of the same kind taken from another individual. At any rate, the protoplasmic substance of moderately malignant epithelial tumors in human beings can be injected subcutaneously in large quantities with no injurious results.

A New Remedy for Ankylostomiasis.—Wijn (*Janus*, 1910, v, 178) in Java has found a new remedy for hook-worm infection. His prescription is: Oil of eucalyptus, 2.5; chloroform, 3.5; castor oil, 40. This dose is taken on an empty stomach in the morning and is to be repeated in two hours. The treatment may be repeated three times in a week, if necessary. A dose of castor oil should be given the day preceding the treatment. In debilitated patients, enemas, though less efficacious, may be employed. No deleterious effects have been noticed from the drug, but drowsiness is frequently a temporary result. In Wijn's experience, a single repetition of the treatment usually suffices to rid the patient of all the parasites.

The Pathology of Addison's Disease.—Porges (*Ztschr. f. klin. Med.*, 1910, lxx, 243) has shown in a previous paper that bilateral extirpation of the suprarenal glands leads to a hypoglycemia in dogs. The adynemia, which is one of the characteristic features of Addison's disease, was found to appear coincidently with the hypoglycemia. He, therefore, determined to study the glycogen content of the liver after the same operation. Dogs were fed on a rich carbohydrate diet for some time and several hours before the operations were given, 100 grams of glucose by the stomach tube. Such a diet leads to a great deposition of glycogen in

the liver, amounting to 7.3 per cent. to 18.69 per cent. as Schondorff has shown. In one of the author's animals whose muscular strength remained good, 3.5 per cent. of glycogen was found in the liver. In the remaining nine animals, the highest glycogen content of the liver following double extirpation of the adrenals was 0.788 per cent., while in five of them the glycogen had entirely disappeared. The muscle glycogen was also absent in two animals. All of the nine animals with low or absent liver-glycogen were markedly asthenic. Porges believes, then, that the loss of adrenal secretion is responsible for the low glycogen content of the liver and the hypoglycemia, and that these two factors in turn account for the marked muscular weakness. In a footnote, the author suggests the advisability of trying a rich carbohydrate diet in patients suffering from Addison's disease.

The Blood of Newborn Infants.—Since one of the arguments against the regenerative and in favor of the degenerative nature of basophilic granules in the erythryocytes has been the frequent failure to find them in the blood of newborn infants and in human embryos, KONIG (*Folio Hæmat.*, 1910, ix, 278) has re-examined the field with the following results: Basophilic granules are found both in newborn infants (4 to 5 per cent. of cases) and in human embryos (twice in six cases). This furnishes a further argument for the regenerative nature of these bodies. Nucleated red cells were found much more constantly, being seen in 92 per cent. of newborn babes examined.

Hemolytic Substance in Schistosomum Japonicum.—YAGI (*Arch. f. exp. Path. u. Pharm.*, 1910, lxii, 156) has sought a cause for the severe anemia which is one of the striking features of infection with Schistosomum japonicum. This trematode is very prevalent in Japan, not only in man but also in domestic animals, such as the ox, dog, cat, etc. The author obtained the parasites from a calf. They were washed and placed in physiological salt solution. To portions of this preparation, ox, cat, and rabbit corpuscles were added. In each instance, the author noted a very weak but definite hemolysis. Since Faust and Tallquist had demonstrated that fatty acids are the active hemolytic agent of Dibothriocephalus latus, Yagi studied the ether extract of Schistosomum japonicum and found that it, too, was hemolytic for the above-mentioned corpuscles, while the ether residue was inert. Lack of material made it impossible to identify the hemolysin, but, by analogy, the author thinks it highly probable that fatty acids or similar bodies derived from Schistosomum japonicum are responsible for the anemia associated with infection with this parasite.

Reticulated Red Blood Cells and Basophilic Granules: Their Clinical and Morphological Significance.—FERRATA (*Folio Hæmat.*, 1910, ix, 253) in an extensive investigation, reaches the following conclusions as to the clinical and morphological significance of reticulated erythrocytes (vital staining) and basophilic granules: (1) Reticulated red cells. The substantia reticula-filamentosa is found normally in a few erythrocytes of the peripheral blood of adult animals; it is seen with much greater frequency (*a*) in the erythrocytes and normoblasts of the bone-marrow, (*b*) in the blood of newborn animals (30 to 40 per cent.) and in still

greater abundance in embryos. Reticulated red cells are also increased in the blood as a result of hemorrhage (traumatic anemia) and in various toxic anemias, especially primary pernicious anemia. The reticulations are plasmatic in origin, since they are formed in normoblasts with intact nuclei and also in the red cells of animals which possess only nucleated red cells. Their presence in the cell indicates its youth and they are, therefore, to be interpreted as a sign of regeneration. (The author might with profit have consulted the American literature, notably the article of Vaughan, *Jour. Med., Research*, Boston, 1903-4, x, 342, in which similar conclusions were reached several years ago). (2) Basophilic granules. Since basophilic granules are missed in the blood of all animal embryos in early embryonic life and first make their appearance when the primitive erythroblasts with relatively small nuclei begin to change into primitive erythrocytes, one must conclude that, in embryos, these granules are visible evidence of the loss of the nucleus and, therefore, a sign of regeneration. Basophilic granules are also found in adult animals in mitotic normoblasts and in the bone marrow, and, being similar morphologically to those in embryos, one must believe that here, too, they are a sign of (pathlogically increased) regeneration. Ferrata believes that these granules are nuclear in origin and, with Pappenheim, assigns them to the parachromatin, while the "jelly bodies" (nuclear particles) are truly chromatic in origin.

Phlorhizin Administration and the Islands of Langerhans.—Van Leersum and Polenar (*Arch. f. Path. u. Pharm.*, 1910, lxii, 266) have investigated the assertion of Lazarus that the prolonged administration of phlorhizin is capable of causing hyperemia, hypertrophy, and hyperplasia of the islands of Langerhans. Suffice it is to say that their observations have failed to substantiate the claims of Lazarus in any particular.

A New Test for Blood in the Urine.—Fleig (*Comp. rend. Soc. de biol.*, 1910, lxix, 192) has made use of the fact that fluoresceine is easily reduced to fluorescine in the presence of oxygenated water and a catalytic agent, as hemoglobin and its derivatives. To prepare the reagent, 0.25 gram of fluoresceine is dissolved in 100 c.c. of a 20 per cent. solution of potassium hydroxide. Ten grams of powdered zinc is added and the whole brought to a boil with constant shaking. The fluorescence disappears on boiling. The solution is filtered cold and preserved in colored bottles with a small amount of powdered zinc. Should fluorescence appear, simple shaking and filtering suffices to clear the solution. For the test in the urine, to 2 c.c. of urine in a test-tube are added 0.25 to 1 c.c. of the reagent and 3 drops of hydrogen peroxide. However little the amount of blood, a beautiful fluorescence is immediately produced. If the tube is shaken the first streaks of fluorescence pervade the whole liquid. The spontaneous fluorescence of some urines can be diluted out of existence without diminishing the resection. The reaction should occur in 3 minutes at a maximum and persist. It is more sensitive than any other test, being positive in dilutions of blood of 1 to 1,000,000. It is practically specific for blood, very simple, easy, and sensitive.

An Experimental and Clinical Study of the Functional Activity of the Kidneys by Means of Phenolsulphonephthalein.—The pharmacological properties of phenolsulphonephthalein suggested to ROWNTREE and GERAGHTY (*Jour. Pharm. and Exper. Ther.*, 1910, i, 579) the possible advantages of its use as a functional test of the kidneys. The studies of Abel and Rowntree had shown that properly prepared solutions of the sodium salt can be injected under the skin without the slightest evidence of an irritative action. The drug can be given also by mouth without untoward effects. The administration of 0.1 gram by mouth is followed by its appearance in the urine in one to one and a half hours; the subcutaneous injection, within ten minutes. The non-toxicity of the substance was definitely established. In the application of the test the time of appearance of the drug is determined, and the amount recovered in a definite period of time. Before each test, 300 c.c. of water is given to insure free urinary secretion, and the bladder is emptied by a catheter. Noting the time, 1 c.c. of the phenolsulphonephthalein solution containing 6 mg. of the drug is injected subcutaneously, the urine is allowed to drain per catheter into a test-tube containing a drop of 25 per cent. NaOH solution in order to show clearly the first appearance of the drug by the pinkish color. After the appearance is noted, the catheter is withdrawn and the patient is instructed to void at the end of the first and second hours. The specimens are measured for their content of the drug by means of the colorimeter. In a series of cases in which the kidneys were normal as far as would be determined, the time of appearance varied from five to eleven minutes; 40 to 60 per cent. of the drug was excreted in the first hour, and 20 to 25 per cent. in the second; 60 to 80 per cent. was excreted in the two hours. The excretion of the drug was independent of the output of urine. In 3 cases of acute nephritis the time of appearance was delayed, and from 4.8 to 44 per cent. was excreted. Neither in these nor in 8 cases of chronic parenchymatous nephritis was there any evidence of increased permeability. Very marked decreased elimination in cases of long standing may be due to secondary sclerosis. The authors suggest a prognostic value of the test in such conditions. In all of ten cases of chronic interstitial nephritis the reduction in the amount eliminated was definite. The graver cases have shown a very small excretion. In cases of enlarged prostate, the authors found the test of more value than the study of the urinary output, total solids, total nitrogen, and urea estimations. A delay in appearance has suggested decreased permeability, but a marked decrease in amount has invariably meant a severe derangement of renal function. Repeated tests will eventually determine the nature, whether a chronic interstitial nephritis or a functional or secondary derangement. In addition, by ureteral catheterization, the test has been applied to the estimation of the function of one kidney as compared to the other. In normal cases, the time of appearance of the drug from the two sides has been almost always the same, from five to ten minutes. The same applies to the amount excreted. When one kidney is diseased, the time of appearance is delayed on the diseased side and the amount excreted is not only relatively but absolutely decreased. The combined output may equal that of two normal kidneys. After nephrectomy in such cases, the remaining kidney eliminates an amount greater than the combined output before operation.

SURGERY.

UNDER THE CHARGE OF

J. WILLIAM WHITE, M.D.,

JOHN RHEA BARTON PROFESSOR OF SURGERY IN THE UNIVERSITY OF PENNSYLVANIA; SURGEON TO THE UNIVERSITY HOSPITAL,

AND

T. TURNER THOMAS, M.D.,

ASSOCIATE IN SURGERY IN THE UNIVERSITY OF PENNSYLVANIA; SURGEON TO THE PHILADELPHIA GENERAL HOSPITAL, AND ASSISTANT SURGEON TO THE UNIVERSITY HOSPITAL.

A New Method of Performing Nephrectomy.—CASPAR (*Zentralbl. f. Chir.*, 1910, xxxvii, 793) says that few conditions have given rise to so many varieties of operation as movable kidney. Caspar has several times had the opportunity of operating the second time on a kidney which had been decapsulated in the first operation. In every such case he found that the kidney was so firmly embedded that it could be separated only with great difficulty from the surrounding tissue. On its posterior surface it was so intimately adherent to the quadratus lumborum, and on its anterior surface with the abdominal wall, that a more ideal fixation could not be desired. With the usual oblique incision in the lumbar region, the layers of the abdominal wall are divided and the kidney exposed surrounded by its fat. The peritoneum with the retroperitoneal tissue are pushed forward as far as possible and the retrorenal fascia divided as far posteriorly as possible where the kidney lies on the quadratus lumborum. It is then shelled out of its fatty capsule. The true capsule is then carefully stripped, beginning with an incision through it along the convex border of the kidney. On the anterior side the separated fibrous capsule is cut away about 1 cm. from the hilum. Only the upper half of the posterior portion is cut away, the remaining lower half being folded and fixed with two catgut sutures to the quadratus lumborum in such a position that at least half of the kidney lies under the ribs. Against and under the lower pole is packed a support of iodoform gauze. The wound is then closed by layer suture, leaving only a narrow opening for the passage outward of the strip of iodoform gauze. This is removed in five to seven days. In fourteen days the wound will be almost healed, and in three weeks the patient can be got out of bed. Caspar has done this operation in 29 cases, 19 times for movable kidney and 10 times for nephritis when it was done to overcome anuria, colic, or hemorrhage. In one of the latter a markedly contracted kidney was found and the patient died. In all the others the kidney became firmly adherent to the surrounding structures and in none did recurrence take place.

Exposure of the Base of the Skull by a Temporary Resection of the Palate.—HOFMANN (*Zentralbl. f. Chir.*, 1910, xxxvii, 817) says that up to the present time the exposure of the base of the skull through the palate has been the least developed of the operations to expose the base of the skull, and that it has been done only by a median splitting of the

soft palate and a more or less extensive resection of the hard palate. To gain a freer exposure Hofmann did the following operation: An incision is made through the mucous membrane transversely from the right premolar across the palate to the left premolar and down to the bone. At right angles to this incision another is made along the alveolar process to the palatopharyngeal arch. The direction of the incision may be reversed according to the site of the tumor to be removed. Over the hard palate the incision passes to the bone and through the whole thickness of the soft palate. The hard palate in the line of the incision is chiselled through, and by means of a periosteal elevator this osteoplastic flap is easily turned to one side. The nasal septum either breaks or can be cut through. The tumor at the base of the skull in Hofmann's case was thus exposed almost in its whole extent and was easily removed. The replacing of the flap was likewise easily accomplished. Two sutures in the transverse and two in the longitudinal portion of the incision sufficed to fix the flap in place. The hemorrhage was naturally slight owing to a preliminary ligation of the external carotid. Hofmann thinks it would still be slight in the absence of such ligation.

Therapy by Bacterins and Tuberculins in Mixed Suppurative Bone and Joint Disease.—WILLARD and THOMAS (*Annals of Surgery*, 1910, li, 761) say that inoculations by bacterins and tuberculins, either alternately or separately, depending upon the condition of the patient, furnish a valuable accessory therapeutic measure in the routine antituberculous hygienic and surgical treatment of mixed suppurative bone and joint disease. Bacterins especially tuberculins, are more potent agents for evil than for good, unless competently administered. Carefully employed, cases do no better than without bacterins, their detention in the hospital is materially shortened, and complications, if they occur, are fewer and less severe. The therapy is superfluous in mild cases where simple operation and time will effect cure; nor is it applicable to neglected cases with prolonged suppuration, characterized by bacteremia, grave sapremia, and amyloid disease. So long as the temperature fluctuates above 100°, autogenous bacterins, obtained by culturing and reculturing the suppuration found to contain variable bacteria from time to time, are administered. Tuberculin inoculations are begun when the temperature falls to 100° or preferably lower. Better results have attended the process of active immunization, where, just as in tuberculin therapy pure and simple, the treatment has been commenced with relatively small bacterial inoculations, progressively increased to the therapeutic limit, rather than by recourse to large dosage, thereby establishing immunity in the former case and in the latter avoiding anaphylaxis. Tuberculin alone in the earliest stage of acute tuberculosis has not proved of noteworthy value, probably due to the fact that the disease progresses too rapidly for the production of immunity, a result which necessarily consumes months. Studious observations of the clinical symptomatology have invariably sufficed to control the inoculations, the opsonic index as a guide proving not only impracticable but frequently erroneous.

Double Dislocation of the Astragalus.—Trenel, Worms, and Doeckel (*Archiv. gén. d. chir.*, 1910, vi, 551) report a case of a complete external dislocation of the astragalus on the calcaneum and the scaphoid and an incomplete dislocation of the ankle-joint, the foot turning inward in the varus position. The patient was a chronic alcoholic. Under chloroform anesthesia the reduction was accomplished by extension, counterextension, and propulsion combined. The foot was drawn downward, and an assistant made counterextension on the leg, while pressure was made directly on the prominent portion of the astragalus, pushing it into place. Two days later the temperature rose, chills occurred, and an increasing dyspnœa set in. There was a cough, with abundant expectoration, greenish and fetid, and other signs of a bronchopneumonia developed, to which the patient succumbed four days after the accident. An autopsy showed the above dislocation and the evidence of the pneumoñia. In a subastragaloid dislocation the astragalus maintains its normal relations with the tibia and fibula, while the calcaneum, scaphoid, and the remainder of the foot are displaced under it. The cause of the dislocation of the astragalus is a very severe force, generally in the young and vigorous, transmitted from above downward against the astragalus, the foot being in a varus position and forced adduction. Reduction should be attempted early, to avoid necrosis of the skin stretched over the prominent astragalus. General anesthesia is necessary to relax the rigid muscles, especially the gastrocnemius. Flexion of the leg on the thigh is very valuable. The fear of ultimate necrosis of the largely detached astragalus should not be regarded as a contra-indication to reduction. The increasing number of operative reductions of total dislocations of the astragalus prove that the nutrition does not become compromised. Pouzat, in 1884, in 31 cases of complete dislocation, showed 19 successful reductions.

Indications for the Different Methods of Operation for Hypertrophied Prostate.—Bensa (*Ann. d. mal. d. org. gén.-urin.*, 1910, i, 1903) says that no one operation can displace all the others. We ought to use several methods to suit the various forms of hypertrophy. His rule is to examine the patient from three points of view. First, he uses cystoscopy, if possible, to fix the form of the hypertrophy. He then determines its nature and consistency by the rectal touch, and takes into account the clinical signs, the hematuria, and the different secretions. Finally he goes over the general condition, and examines the lungs, heart, and kidneys. According to the form of the hypertrophy, its nature, and the general condition of the patient, the choice of operation is determined. Total transvesical prostatectomy is applicable to fibro-adenomatous forms involving the lateral lobes, in patients still young and little infected. It should be done in two stages in those infected temporarily. Notwithstanding our progress in technique, patients still die of hemorrhage and at times of infection, which is more easily avoided. Whatever the method of operation and the habits of the operator, the mortality remains about 10 to 15 per cent. Partial transvesical prostatectomy is indicated for pedunculated median lobes. Perineal prostatectomy is preferable for the suppurating forms, because of the facility of drainage, and sometimes for the low forms of hypertrophy. The Bottini operation is appropriate for soft hypertrophy of the lateral lobes (which can be

enucleated with difficulty if at all), hypertrophies of the median lobe in the form of a bar, and for stenoses of the vesical neck. It is also indicated when the patient refuses the operation by the abdomen and, finally, when, because of his serious condition, he does not seem able to sustain any other serious operation. This operation is very rational in that it removes only that which obstructs the posterior urethra and neck of the bladder, and, almost without danger, relieves the retention of urine. Suprapubic cystotomy should sometimes be associated with the deep cauterization of the obstruction at the neck of the bladder or of the prominent lobes, in the bleeding cases, or in the infected. Sometimes it may be employed alone to keep the bladder healthy and clean, by interposing a means of discharge between the lower and the upper urinary paths. It constitutes then the last resource in those patients in whom all the ramifications of the urinary apparatus are distended and infected.

Post-traumatic Osteitis of the Carpal Scaphoid Tending to a Spontaneous Fracture.—PREISER (*Zntrlbl. f. Chir.*, 1910, xxxvii, 929) calls attention to what he considers to be a not very rare post-traumatic osteitis of the carpal scaphoid bone, of which he has seen 6 cases. As a rule, the condition pursues the following course: After a fall upon the hand there develops, with swelling and pain, usually a slight limitation of motion, and tenderness over the scaphoid, especially in the tabatierre anatomique. The Röntgen rays showed in five cases a more or less round or elongated clear space, which often involved the whole of the scaphoid. A primary fracture does not seem to precede it. Gradually the defect increases, the walls break through, and the scaphoid finally gives about the same picture as a recent fracture. The clinical course is very chronic, the disturbances remaining only in mild degree. The condition probably originates in a primary tearing of the dorsal ligament between the scaphoid and semilunar bones, which leads to a disturbance of the nutrition of the central portion of the scaphoid, with resulting rarefying osteitis, which shows in the skiagram as a visible defect and finally causes a spontaneous fracture. The scaphoid is almost entirely surrounded by joint surfaces and is chiefly nourished, as Lexer's injection experiments show, by the bloodvessels in the scaphoid-semilunar ligament. The rarefaction appears very soon. In one case in two days after the accident there were visible in the skiagram three round defects, which, in spite of fixation, in ten days had united with the formation of a transverse clear space in the bone. Then followed, however, as the later skiagrams and the complete clinical restoration demonstrated, a healing in of the defect. Probably by this time the other small vessels to the bone have reëstablished the nourishment of which it was deprived.

The Treatment of Gangrenous Hernia without Immediate Removal of the Incarceration (Entero-anastomosis above the Hernial Canal).—HESSE (*Zntrlbl. f. Chir.*, 1910, xxxvii, 1033) says that it is gradually becoming recognized that the most desirable result is obtained from a primary resection of the incarcerated portion of the intestine and the immediate renewal of the intestinal current, by suture of the divided ends of the bowel. This exposes the patient to the danger, however, of a septic

peritonitis and to the risk of an extensive and prolonged operation in his weakened condition. When, however, the hernial contents are foul or suppurative, in consequence of the disturbances in the intestinal wall, or there is a perihernial phlegmon, the danger of peritonitis may be excluded by performing first an entero-anasotmosis, and leaving the separation and removal of the gangrenous and resected bowel to the end of the operation. If a perihernial phlegmon is diagnosticated, it should not be attacked first; or if the incision over a strangulated hernia exposes a threatening or actual perforation of the gangrenous loop of intestine, this part of the operation should be stopped at once. Then with slight elevation of the pelvis an incision should be made parallel with and 3 cm. above Poupart's ligament, and should extend into the abdomen. The afferent and efferent limbs of the incarcerated intestine will readily be recognized. If they are in good condition, a lateral anastomosis 5 to 6 cm. wide is made between the two limbs, far enough away from the incarceration to permit careful handling of the intestine without pulling on the incarceration, which should be covered with gauze, and to permit at a later period a resection of the excess of bowel and a closure of the open portion. The corresponding mesenteric surfaces are approximated by a few sutures to prevent the formation of an opening which may lead to a later ileus. The laparotomy wound is closed completely or partially. In the latter case a strip of gauze is introduced to act as a protection from the inner opening of the hernial ring. Now for the first time the phlegmonous area is opened extensively and the foul contents of the hernial sac and gangrenous intestine are evacuated. The strangulation is not relieved nor is the incarcerated intesine in any way pulled upon. A suitable dressing isolates the two wounds from each other.

Hemorrhagic Glossitis in Hemophilia.—UMBREIT (*Deut. Ztschr. f. Chir.*, 1910, cv, 608) reports the case of a man, aged thirty years, who had no family history of hemophilia, but whose life had been threatened several times from hemorrhage due to the extraction of teeth and similar trivial wounds. On December 5, 1909, a red spot appeared on the tip of his tongue, which rapidly became a painful swollen mass, giving rise to a marked dysphagia and dyspnœa. He sought admission to the hospital for relief on the same day, when the tongue protruded 2.5 cm. in front of the separated teeth, was of a deep bluish-black color, stony hard, and immovable, and was 3.5 cm. thick. The floor of the mouth and the region between the chin and the hyoid bone were infiltrated with blood. The posterior part of the tongue showed no changes. Breathing was difficult and swallowing impossible. The temperature was 37.5° C. and the pulse 120, moderately strong, and regular. The internal organs were normal and the urine free of sugar and albumin. After puncture, painting with adrenalin, and other such measures, without success, on the following day, notwithstanding the danger of a possible severe hemorrhage, an incision was made in the floor of the mouth. A forceps was introduced into a mass of coagulated blood about the size of a walnut. No hemorrhage occurred, and the wound was tamponed. On the following day the removal of the tampon was followed by the trickling escape of a thin fluid blood stream, which continued for five days, in spite of all measures to stop it, until a very

marked anemia developed. The day before this the temperature rose to 39.2° C. and the pulse to 164, which became very weak and irregular. An extensive hematoma developed under the chin, the skin here became dark blue, the lymph nodes much swollen, and the patient complained of much headache. On the tenth day the swelling subsided slightly, the skin became red, and necrotic tissue escaped from the wound under the tongue. The secretion again became bloody, and the bleeding did not cease for three days. Gradual improvement set in and continued until, on January 13, the patient was discharged cured.

A New Method of Closing the Intestine.—KLAPP (*Deut. Ztschr. f. Chir.* 1910, cv, 559) closes the divided end of the intestine by first rolling it over on itself with a suitable forceps. The portion to be divided is first crushed by a proper pressure forceps and then seized with a long, narrow, and tightly closing intestinal clamp. It is now divided close to the forceps, preferably with a thermocautery, and then rolled on the forceps until there is a good serous contact between the rolled portion and the neighboring intestine, when a sufficient number of Lembert sutures are placed to fix it in this position. Finally, the branches of the forceps are opened, separated from each other, withdrawn individually, and the small opening through which they were removed is closed by a few sutures. When carried out on the lower animals, as in performing the Billroth II operation for resection of a portion of the stomach, and the duodenal end had been turned analward and closed, strong distention of the duodenum with air or water permitted no leakage at the site of the closure. When the distention was increased more and more it was found that not the closed end, but other parts of the bowel gave way. In actual practice, he employed the method twice in the Billroth II operation, three times in a resection of the large intestine, and frequently with the appendix.

Treatment of Wounds of the Heart.—KIRCHNER (*Annals of Surgery* 1910, lii, 96) says that the heart may be manipulated without serious injury to the organ, and is amenable to surgical interference and procedure. Hemopericardium with heart tamponade is a serious complication, and demands prompt drainage of the pericardial sac. In suspected injuries to the heart the wound in the chest should be carefully explored so that the extent of the injury may be determined, and in cases of doubt exploratory pericardiotomy is indicated. Small wounds of the heart may heal spontaneously, but in all cases where hemorrhage from the heart exists, the wound should be promptly sutured. In operations upon the heart, when pulsations suddenly cease massage of the heart and artificial respiration should be tried as aids in resuscitation. With a minimum amount of anesthetic the healthy lung is capable of performing its function, though to a less extent, even when the pleural cavity is exposed. Time is an important factor in injuries to the heart, and an early diagnosis should always be made. The immediate treatment should be directed to the control of hemorrhage and shock. The chief remote complications result from infections of the pericardium and pleura. In treating injuries to the heart, the surgeon should have in mind a definite plan of attack, and the kind of a flap to be used in approaching the heart should be determined by the nature of the wound in the chest.

THERAPEUTICS.

UNDER THE CHARGE OF

SAMUEL W. LAMBERT, M.D.,

PROFESSOR OF APPLIED THERAPEUTICS IN THE COLLEGE OF PHYSICIANS AND SURGEONS, COLUMBIA UNIVERSITY, NEW YORK.

The Use of Sour Milk in the Treatment of Constipation.—LUKE (*Practitioner*, 1910, lxxxiv, 653) has had good results with the use of sour milk in constipation. His method of administering it is as follows: Patients usually take their first glassful at breakfast in the place of porridge. It is convenient to turn the milk out into a cold pudding plate and sprinkle the curd with sugar, or with a mixture of cinnamon and sugar. Some patients enjoy the slightly acid, sharp taste, and find it more agreeable and appetizing without any added sugar. A brown meal biscuit goes well with it. At lunch the patient takes the curd instead of soup, and at dinner to replace any sweet pudding or pastry. This makes a convenient distribution and prevents the patient feeling that the extra bulk of food is embarrassing. Some patients who are on a purin-free diet and do not take afternoon tea find a glass of curd acceptable at 4 P.M., especially in hot summer weather. Luke has never found patients to suffer any material inconvenience or digestive discomfort which was traceable to this diet. If any flatulence or discomfort is experienced for a day or two, the amount of milk taken should be reduced and gradually increased once more as the patient gets accustomed to it. The time for which the milk is taken should never be less than six weeks, if it agrees at all, and it may be quite well continued for six months, and afterward taken occasionally as an article of daily diet.

The Soy Bean as an Article of Diet for Infants.—RUHRÄH (*Jour. Amer. Med. Assoc.*, 1910, liv, 1664) advocates the use of the soy bean as an article of diet for infant feeding and diabetics, on account of its large percentage of protein. The soy bean, sometimes incorrectly called the soja bean, is a staple food among the Chinese and Japanese, and probably accounts in part for the small amount of other nitrogenous food taken by them. Rührah thinks that this general and continued use of the bean demonstrates conclusively that the protein of the bean may be utilized in the body in place of that of other vegetables and of meats. The soy bean may be cooked in the form of soups, gruels, and porridges, or may be eaten raw as a vegetable or salad. He does not think, however, that the soy bean will ever replace meat in ordinary diet, but that it will be found of value as a source of protein when meat is contraindicated and especially when, as in diabetics, the patient tires of meat. He also believes that it is of great value in difficult feeding cases in infants. As soy beans are not available in most markets, and as their cooking is a tedious process, Rührah had a flour manufactured from the bean. The analysis of this flour showed a protein content of 44.64 per cent. The percentage of protein was almost one-third greater than the

percentage in the whole beans, due to the removal of the coarse fibrous hulls. Each ounce of this soy gruel flour yields about 13 grams of protein and 120 calories, and it can be used (1) as a gruel, (2) in broths, (3) in making biscuits or muffins. It may also be mixed with cereals, barley jelly, cream of wheat, and similar foods. Rührah gives the method of preparation of gruels, broths, and muffins from this soy-bean flour in his article. He also discusses the indications for its use in difficult infant feeding cases, and believes that the soy-bean flour solves the difficulty of supplying sufficient protein in a form in which it can be digested and assimilated by infants and young children.

The Physical Treatment of Bronchial Asthma.—HOFBAUER (*Med. Klinik*, 1910, vi, 894) is of the opinion that incandescent light baths are of great value in the treatment of bronchial asthma. The electric light baths induce an active hyperemia of the skin, thus reducing the congestion of the bronchial mucous membrane with a consequent increase in the size of the lumen of the bronchus. Hofbauer thinks that their good effects are largely due to the prolongation of the expiration induced by the heat. This effect is supplemented by breathing exercises, to lengthen the period of expiration, which the patient accentuates by sounding a musical note. Hofbauer has cured a number of patients by these breathing exercises, the cure remaining complete for over two years.

Vasotonin: A New Remedy for Lowering Blood Pressure.—MÜLLER and FELLNER (*Ther. Monatshefte*, 1910, xxiv, 285) report both animal experiments and clinical observations concerning the effect of vasotonin upon blood pressure. Vasotonin is a combination of yohimbin and urethane. Müller conducted the animal experiments, and found that vasotonin lowered the blood pressure by dilating the peripheral blood-vessels. He could determine no depressant action upon the heart muscle. There was no depression of the vasomotor centre, and no effect upon the respiration occasioned by its use. Fellner relates his clinical results obtained in 30 cases of increased arterial tension. He gave vasotonin subcutaneously in doses of 1 c.c. The injections were given in some cases daily, in other cases every other day. The course of treatment comprised from 20 to 30 injections. He found that the remedy consistently produced a fall of blood pressure with a marked improvement in the subjective symptoms. Thus, for instance, there was immediate relief in the milder cases of angina pectoris, and in cardiac and bronchial asthma. The severer cases of angina pectoris required a longer period of treatment, but also showed distinct improvement. No unpleasant symptoms occurred as a result of the injections except some feeling of fulness in the head, but not equal to that caused by the nitrites or nitroglycerin.

The Treatment of Delirium Tremens with Veronal.—VON D. PORTEN (*Therapie d. Gegenwart*, 1910, li, 270) compares the results obtained by the treatment of delirium tremens with veronal with those obtained by the use of chloral hydrate and bromides. There were 396 cases treated with chloral and bromides. The treatment in these cases consisted in the administration of from 15 to 45 grains of chloral hydrate,

given usually only on the first day, but occasionally repeated on the second day. The chloral was often combined with 15 grains of bromide given every two hours. Of these cases, 77, or 18.1 per cent., developed a frank delirium and 36 died, thus giving a mortality of 9 per cent. The number of cases treated with veronal was 264. These patients received 15 grains of veronal in warm tea, which dose was repeated in one or two hours. The amount was usually enough to make the patient quiet and insure a deep, quiet sleep. If this result was not obtained, a third dose of 15 grains was given after five hours. The author notes also that in the severest cases it was necessary to give as much as 60 grains during the first twelve hours before the desired result was obtained. He has never seen the slightest untoward effects upon the part of the pulse or respiration from this dosage. Of the cases so treated, only 15, or 5.6 per cent., developed a frank delirium, while the mortality fell to 3.4 per cent., as compared with 9 per cent. in those treated with chloral and bromides. The author also notes that the period of delirium averaged much less in the cases treated with veronal.

Bronchial Asthma in Relation to the Exudative Diathesis.—Von Strümpell (*Med. Klinik*, 1910, vi, 889) states that bronchial asthma should be included in the group of affections distinguished by an irritability of the nervous system and a tendency to exudative processes in various parts of the body. He mentions as members of this group, urticaria, eczema, angioneurotic œdema, intermittent swelling of the joints, and nasal and intestinal catarrh. He considers that membranous colitis, with the secretory disturbance of the mucosa accompanied by a spasmodic contraction of the muscular coats of the intestine, is closely related to bronchial asthma. Membranous colitis may therefore be entitled "intestinal asthma." Von Strümpell thinks that the nervous factors explain the benefit derived from atropine, adrenalin, and other remedies advised in asthma. He has had the best results in treating bronchial asthma by the use of the alkaline iodides, and the systematic application of incandescent electric light baths. He believes that both serve to correct the overproduction of mucous and so lighten the severity of the attacks. These must be supplemented by dietetic restrictions to prevent overfeeding.

Gout.—Foster (*Yale Med. Jour.*, 1910, xvi, 477) draws a sharp demarcation between the various symptoms which are commonly attributed to "goutiness" and the clinical entity of gout. The symptoms which are assigned to goutiness are due to other causes, as these cases usually present none of the characteristic stigmas of gout. In the treatment of acute attacks of gout Foster considers that the prime object to be secured is the comfort of the patient. To this end he advises the use of wet dressings or protection of the inflamed joints from pressure. If the pain is severe, opium must be employed. Colchicum is the only drug that he considers of any value at this stage of the disease, but caution should be exercised that the digestion is not deranged by its prolonged use. During the febrile interval the food should be limited to milk and gruels. Convalescence may be hastened by hot baths with gentle manipulation of the affected joints while in the bath, and light massage afterward. Foster thinks that the result secured in

treating individuals who have had one or more attacks of gout must be mostly through the diet and mode of life. He gives the following as fundamental principles: Build up the cachectic and undernourish the obese; compel the patient to take all the exercise his strength permits; avoid sudden changes in diet or habits; give as much attention to the digestive function of the patient as to the purin content of his diet. All of the dietaries that have been advised for gout in the last two hundred years present two features; an endeavor to restrict the total amount of food taken and to lessen the chance of gastro-intestinal disorders. Foster believes that there is no excuse for overfeeding with our more accurate methods now of determining the actual food demands. The amount of meat that should be allowed each patient must be determined by obtaining the excretory capacity of the patient. This can be done only by accurate urine analysis. Whether the elimination be prompt or restricted, a period of four or five "purin-free" days in each month is often advisable. Foster thinks that little benefit is derived from the use of drugs. No drug is known to have any influence on uric acid metabolism, much less the power of dissolving the deposits of sodium urates in the joints. The claims made for some drugs, such, for example, as piperazin and quinic acid, rest on test-tube experiments that are not borne out when applied to living tissues. The use of alkalies, first advocated by Boerhaave, has not now the repute it formerly held. Von Noorden asserts that alkalies cause a retention of uric acid and precipitate attacks. The salts of lithium are entirely useless. The only drugs which Foster considers to possess any value are the salicylates, which are occasionally given to relieve pain, and the iodides. Hydrotherapy is of great help in many cases. The "cures" at resorts are of undoubted benefit, but when the patient cannot leave home much can be accomplished by means of hot air and steam baths with massage.

Further Observations upon the Practical Application of Sodium Chloride in the Treatment of Epilepsy.—Ulrich (*Münch. med. Woch.*, 1910, lvii, 1173) advocates sodium chloride for the treatment of the symptom complex known as bromism occuring as a result of long-continued use of the bromides. He says that sodium chloride seems to have a physiological action directly antagonistic to that of the bromides. He believes that sodium chloride is able to cure promptly and certainly the motor, sensory, and psychic symptoms of acute bromism. All skin affections disappear promptly with the administration of sodium chloride. Ulrich has also noted that sodium chloride is able to incite epileptic attacks in old epileptics who have been so saturated with bromides that they have reached a state bordering on insanity. He believes that after these induced convulsions these patients are in a much better mental condition than before. Ulrich is firmly convinced that sodium chloride is far superior to all of the present remedies for bromism.

PEDIATRICS.

UNDER THE CHARGE OF

LOUIS STARR, M.D., AND THOMPSON S. WESTCOTT, M.D.,
OF PHILADELPHIA.

Epigastric Pain as a Complication of Mumps.—Supplementing Dr. Godfrey's article on epigastric pain as a complication of mumps being due to pancreatic metastasis, CECIL REYNOLDS (*Brit. Med. Jour.*, 1910, ii, 352) suggests that this condition is due to the organisms of mumps or secondary organisms growing on a dry tongue and being capable of producing acid from sugars, setting up a fermentation of carbohydrates when swallowed, and thus causing a pylorospasm. He has seen this condition in mouth breathers from whatever cause. Edward Penny reports seeing many cases of epigastric pain with vomiting in the last stages of mumps. Out of 20 cases in one school, 10 followed this course, and all showed tenderness to pressure over the pancreas. Fox reports a similar case: on the fifth day of mumps a boy developed fever, epigastric pain, and vomiting, and a deep-seated swelling was felt in the epigastric region. There was no sugar in the urine, and the boy recovered.

Hereditary Lues and the Wassermann Reaction.—PAUL MULZER and WALTER MICHAELIS (*Berl. klin. Woch.*, 1910, xlvii, 1402) have tested the clinical worth of the Wassermann reaction, and especially a modification of this reaction in which a smaller amount of serum is used for work on children with hereditary syphilis. They use about 0.05 c.c. of the serum taken from about ten drops of blood; and they used it only in syphilitic children or in suspected cases. Out of 44 children, manifestly luetic, with lesions on the skin and mucous membrane, they found a positive reaction 42 times and a negative reaction twice. In early congenital lues they found a positive reaction in 95.6 per cent. One child of two months gave a negative reaction three weeks before the syphilitic outbreak (the parents being syphilitic). With the first development of the disease the child gave a positive Wassermann reaction. Another child with two syphilitic sisters gave negative reactions until after the development of a coryza. A seven months' premature infant, whose father had syphilis, gave a positive reaction, but showed no physical signs of the disease. A premature infant whose mother had syphilis and gave the positive Wassermann reaction showed no physical signs and a negative reaction. In 44 children with latent syphilis, with no acute symptoms or none since a previous treatment, 26 showed positive and 18 negative reactions. More than half of the latent hereditary syphilitic children show positive reactions. Out of 73 children suspected of syphilis and suffering from anemias, enlargements of the glandular system, spleen, or liver, poliomyelitis, and periostitis, 18 positive reactions were found. In 19 such cases only 5 showed a negative reaction after treatment with protiodide of mercury (gr. $\frac{1}{6}$ to $\frac{1}{8}$) and inunctions. Out of 22 mothers of syphilitic children, 21 knew of no syphilitic infection, but 17 of them gave positive reactions; 15 had children

under one year old and two had older syphilitic children. Out of 12 apparently healthy sisters of syphilitic children, 5 gave positive reactions. In infants and children with clinical signs of hereditary syphilis, from 90 to 100 per cent. will show positive Wassermann reactions. Children with manifest lues react like a syphilitic in the secondary stage, 96 per cent. positive. Positive reactions appear first with the onset of the manifest syphilitic symptoms. Latent syphilitic children react as adults do in the early latent period. It is more difficult to change a positive to a negative reaction by anti-syphilitic treatment in children than in adults. Mothers of syphilitic children show, in the greater majority of cases, a positive reaction (83 per cent.). The later children born of syphilitic parents, but having no symptoms of syphilis, show negative reaction.

Chlorosis.— Paul Groag (*Münch. med. Woch.*, 1910, lvii, 1596) supplements the investigations of Morawitz on chlorosis. In his own cases it was not so much the subjective symptoms found in light chlorosis and neurasthenia, such as exhaustion in the morning, lack of desire for exertion, slight headache, as the striking pallor of the face and mucous membranes, which led him to suspect chlorosis. In most of the cases he found a systolic murmur in the heart and accentuated pulmonic second sound. The blood examination always showed the normal number of red blood corpuscles, and hemoglobin from 90 to 95 per cent. In all the cases treatment with iron was followed by the best results, relieving not only the subjective symptoms, but causing a disappearance of the pallor. He agrees with Morawitz in calling these cases chlorosis, with a normal hemoglobin percentage, but an increase in the entire blood quantity. In regard to the etiology, he adds that almost all of his patients gave a history of masturbation or interrupted coitus. Since almost all authorities mention the genital functions in relation to chlorosis, he believes that this association is important, especially so since most of the before-mentioned symptoms of chlorosis are found in cases of masturbation in young men. It is possible that a blood condition analogous to chlorosis, only milder, occurs at puberty in the male sex, in which the number of blood cells and the hemoglobin is normal, but the entire blood quantity is increased.

Vincent's Angina.—J. D. Rolleston (*Brit. Jour. Child. Dis.*, 1910, vii, 299) presents a study of 32 cases of Vincent's angina. This condition presents a faucial lesion usually unilateral, deep ulceration of the tonsil and adjacent structures, a peculiar fetor, and enlargement of the corresponding lymph glands. It is associated with a fusiform bacillus and a spirillum described by Vincent in 1896. This form of angina is uncommon. Out of 3047 cases of angina, only 95 were diagnosticated as Vincent's form. It averages from 0.5 per cent. to 0.9 per cent. in all cases of sore thorat. It is relatively rare in adults, except those working in dissecting rooms or among cadavers. It is most frequent up to the tenth year. It is feebly contagious and is usually transmitted by vessels used in common. It is commonest in spring and rarest in autumn. While Vincent emphasizes previous ill health and oral sepsis as predisposing factors, these were not prominent in this series of cases. An ulcerative and membranous form of

the disease are distinguished, but Rolleston claims the ulcerative is a later stage of the membranous. It often stimulates diphtheria, but the adenitis is not as severe and suppurative adenitis is unknown. The fetor is characteristic and different from that of diphtheria. There is a marked disproportion between the severity of the local and constitutional symptoms, the latter being light and short. The throat heals rather slowly, however, the average time in this series being eighteen days. The co-existence with diphtheria is not common, but does occur, and antitoxin in these cases seems beneficial. When associated with syphilis, mercurial treatment materially assists resolution. The disease is usually primary, prognosis favorable, and complications are infrequent. The treatment consists of local swabbing with undiluted tincture of iodine. A solution of chlorate of potash and myrrh is used for the fetor and a preparation of powdered methylene blue is useful for the ulceration.

Brudzinski's "Neck Sign" and the Contralateral Reflex in Meningitis.—JOHN LOVETT MORSE (*Archives Pediat.*, 1910, xxvii, 561) has investigated Brudzinski's claim that the "neck sign" and contralateral reflex is not found in healthy children, or in any cases of disease except tuberculous or epidemic meningitis. The contralateral reflex is a concomitant reflex of the leg on one side when passive flexion of the leg on the other side is made. The motion of the other leg is extension instead of flexion at times. The "neck sign" consists in passive flexion of the head forward, the other hand being pressed on the chest. In meningitis this will cause flexion of the legs at the hips and knees and a marked flexion of the legs on the pelvis. Greco and Zaimvosky have investigated the contralateral reflex in a large number of cases, and entirely agree with Brudzinski's claim for its value. The "neck sign" was tested in 42 cases of meningitis, where it was positive in 97 per cent., the contralateral reflex being positive in 66 per cent., Kernig's sign in 57 per cent. and the Babinski in 50 per cent. of the cases. Morse examined 400 children, 90 of whom were well and the others suffering from diseases not of the nervous system or meningitis. Neither the "neck sign" nor the contralateral reflex was elicited in any instance. This corroborates Brudzinski's claim. Morse also examined 11 cases of tuberculous, 5 of cerebrospinal, and one of pneumococcic meningitis for the presence of these two signs. The ages varied from six months to eleven years. In the cases of tuberculous meningitis the results were not constant. The contralateral reflex was usually absent; when present it was identical. The "neck sign" was found more often, but was never constant. In the cases of cerebrospinal meningitis the contralateral reflex was never found, but the "neck sign" was positive much more often than in the tuberculous variety, and when present flexion always occurred in both legs. In the case of pneumococcic meningitis the contralateral reflex and the "neck sign" both were present on one side, the other side being paralyzed. Morse concludes that the two signs are never present in healthy infants and children or in those ill with diseases other than those of the nervous system, and that they are almost never present in diseases of the nervous system outside of meningitis. Of the two, the "neck sign" is found more constantly. Their presence in acute disease is strong evidence of meningitis, but their absence does not exclude meningitis.

OBSTETRICS.

UNDER THE CHARGE OF
EDWARD P. DAVIS, A.M., M.D.,
PROFESSOR OF OBSTETRICS IN THE JEFFERSON MEDICAL COLLEGE, PHILADELPHIA.

Gallstones during Pregnancy and the Puerperal Period.—PETERSON (*Surg., Gyn., and Obstet.*, July, 1910) reviews the literature on this subject and reports a case operated upon by Nancrede at six months' pregnancy. Death followed in this case, from postoperative hemorrhage and exhaustion. He finds that gallstones are most commonly met with during pregnancy, in patients between the ages of twenty and thirty-five. Pregnancy undoubtedly conduces to the formation of gallstones. Pregnant patients suffer an attack of pain and distress usually about the fifth month, when the uterus is approaching the level of the umbilicus and beginning to crowd the intestines against the liver. In puerperal cases symptoms usually manifest themselves during the first seven days of the puerperal period. Such patients suffer from chills and fever with jaundice. Sixty per cent. of pregnant patients suffer more or less from jaundice. In the puerperal period the frequency is 10 per cent. Among pregnant patients operated upon the mortality was 13.04 per cent.; in the puerperal period the mortality was 11.01 per cent. In pregnant patients more than one-half of the gallstones were found in the bladder, and this was true of the puerperal cases. There is no reason to believe that operations upon the gallbladder are more apt to interrupt pregnancy than are other abdominal operations. If the patient is in good condition, operation should be deferred until the child is viable, lest premature birth be induced. The diagnosis of the condition in pregnancy and the puerperal state is not especially difficult, and jaundice will be found a valuable symptom.

Vaccine Therapy.—At the recent meeting of the American Gynecological Society (*Surgery, Gynecology, and Obstetrics*, July, 1910) the committee appointed to investigate the employment of vaccine therapy in gynecology and obstetrics, composed of Dr. J. Whittridge Williams, Dr. Edward B. Cragin, and Dr. Franklin S. Newell, reported essentially as follows: The evidence at present available justifies tentative conclusions only. To produce active immunity opsonins undoubtedly have influence. Unfortunately the practical value of the opsonic index is questionable because it is technically difficult and is subject to great variations. Against typhoid, cholera, plague, and dysentery immunization by vaccines is a well-established prophylactic measure. In local infections by the staphylococcus or tubercle bacillus, vaccine therapy is valuable, and less so in local infections with other germs. In acute general infections the value of this method of treatment has yet to be proved. Vaccine therapy is a valuable adjunct and occasionally curative in chronic gonorrhœal arthritis and urethritis. It is also valuable in the vulvovaginitis of children, but does not always succeed. The bacteriuria of infections of the urinary tract by the colon bacillus is

not relieved by vaccine therapy. The symptoms seem sometimes considerably lessened. The scanty reports concerning pyelitis and pyelonephritis in pregnancy do not indicate that this method of treatment is more successful than the usual methods employed. In some cases of endometritis after curetting vaccine therapy seems to be of benefit. There is not sufficient evidence concerning its use in pelvic inflammatory disease to warrant conclusions, but in chronic cases after operation it might prove useful. As the ordinary localized puerperal infections, irrespective of the nature of the infecting germ, tend to spontaneous cure, the use of vaccine therapy must be limited to acute general infections. Here the method appears to be of little value at present, but the most that can be said of it is that it does not produce harm.

Bilateral Tubal Pregnancy with Coincident Development.—FINDLEY (*Surgery, Gynecology, and Obstetrics*, July, 1910) has collected from the literature 28 cases of bilateral tubal pregnancy, in many of which the proofs as to the nature of the condition are not conclusive. But 8 are unquestioned. He adds the case of a nullipara who had disturbed menstruation, hemorrhage, and pelvic pain. The abdomen became distended and a boggy mass filled the pelvis. The uterus was but little enlarged and crowded forward. At operation the peritoneum was inflamed and several pints of fetid blood clots were in the lower abdominal cavity. Both tubes were removed, and on the left side was a tubal abortion, and on the right a blood mole at the ampulla of the tube. Death followed eight hours after the operation from exhaustion and shock. It was thought that the escape of the ovum and blood through the abdominal end of the left tube and of its twin ovum through a rent in the ampulla of the right tube were probably simultaneous. Placental tissue, decidua, and chorionic villi were found in both tubes. No fœtus was found. The ovaries were cystic and bound by adhesions.

When Shall Operation Be Done for Puerperal Septic Infection.—POLAK (*Surgery, Gynecology, and Obstetrics*, July, 1910) reports the results of 200 cases of septic infection as follows: In 72 extensive exudates, suppuration was a late result. The earliest abscess occurred on the nineteenth day, the latest on the eighty-second day. In 3 opening was made by an extraperitoneal incision above Poupart's ligament. Four were opened by vaginal section. In 65 cases the exudate was completely absorbed under the influence of time, rest, and baking with dry heat. Polak believes that a local focus, postpartum, should not be disturbed so long as the patient improves, unless there develops definite evidence of a localized collection of pus. In none of these cases was the application of heat employed until the acute process was in abeyance. There were 5 cases of ruptured uterus, 3 of which had been packed in dirty tenements for the control of hemorrhage, and were therefore considered septic and radical operation performed. Of the 5 cases, 3 made complete recoveries after complications, 2 died, although operation was performed. One case of septic purulent peritonitis following criminal abortion, died, although the peritoneal cavity was drained. The fourth fatal case was admitted in a dying condition and expired a few hours after a Pryor operation. One case died of acute gastric dilatation 52 days after confinement, and eight days after

an abdominal exploration and vaginal drainage for a collection of pus in the right broad ligament. A sixth case died from septic endocarditis and general infection forty-seven days after confinement. Streptococci were repeatedly demonstrated in the blood, and vaccine and supportive treatment failed to benefit the patient. This case had been curetted twice by local physicians. In the 200 cases there were 6 fatalities. The Pryor operation, or isolating the uterus and pelvic peritoneal cavity from the general peritoneal cavity, by the use of gauze packing, was performed twelve times. Polak believes it to be valuable in early pregnancy, where the sigmoid assists in shutting off the pelvic cavity. There were 46 cases of putrid endometritis, all of which recovered with disinfection of the uterine cavity with gauze soaked in iodine. Seventy-two cases had exudates, 63 of whom had been curetted before admission. Suppuration occurred seven times in these cases. There was no mortality. Pelvic cellulitis was present in 16 patients, in whom there had been extensive cervical lacerations. In one case suppuration occurred with fatal results. There were 17 cases of streptococcic septicemia, and 10 cases of bacteremia, with one death from septic endocarditis. There were 2 cases of pyemia with multiple foci, both of whom recovered; 126 suffered with pelvic peritonitis, all of whom recovered with vaginal drainage. Five cases of uterine and femoral thrombophlebitis recovered with protracted convalescence. In 3 cases of septic purulent peritonitis, 1 died and 2 recovered. In these the treatment consisted in ordinary abdominal incision, tube drainage with aspiration of the tubes every three hours, the high Fowler position, the instillation of salt solution, and gastric lavage. Starvation was practised until the patient began to improve. There was one case of intramural abscess in the uterus, which recovered with drainage. The general conclusion of this series of cases is, that operation must be determined by the merits of each individual case only; that so long as the patient is improving, interference should not be practised; that care must always be taken not to break down the protective barriers which Nature establishes in these cases.

Early Rising in the Puerperal Period.—VELITS (*Zent. f. Gyn.*, No. 25 1910) gives the results of his investigations in 496 puerperal cases, many of them being in the hospital during pregnancy; 68 per cent. were allowed to get up early after confinement; of those coming into the hospital at the time of confinement, 59 per cent. The latest period was seven days after labor and the earliest five. Afterward patients were allowed to get up earlier, and two left their beds the first day after confinement. At first this was limited to patients who had had normal labors, but afterward those who had had operations, even of some severity, were also included. In 174 operative cases 70 got up within a short time after the operation. In 84 patients having sutures inserted for lacerations of the vagina and perineum 33 who got up early had healing by first intention, their wounds finally closing without complications. In these cases aluminum bronze wire was used for suture. It was observed that involution went on rapidly in these cases, that a bloody lochial discharge ceased early, and that bronchial secretions in general did not endure as long as when patients remained in bed. The use of the catheter was avoided and the bowels acted more regularly and earlier. Thrombosis and embolism were not observed.

GYNECOLOGY.

UNDER THE CHARGE OF

JOHN G. CLARK, M.D.,

PROFESSOR OF GYNECOLOGY IN THE UNIVERSITY OF PENNSYLVANIA, PHILADELPHIA.

Experimental Researches upon the Nervous Reflexes from Different Organs and Peripheral Nerves upon the Uterus.—The reflex relations existing between the uterus and other organs of the body, especially the breasts, gastro-intestinal canal, nose and bladder, have been commonly recognized by clinicians, based purely upon clinical observation or the acceptance of empirical facts as handed down from generation to generation. Therefore but little effort has been made to prove by actual experimentation the truth or fallacy of these observations, or to determine the pathways by which such reflexes are carried. In a most interesting and instructive article, E. KEHRER (*Archiv für Gynäk.*, 1910, xc, 169) reports in full the results of his painstaking and difficult experiments concerning this important subject. Following a concise description of the general technique employed, he details his methods of procedure upon the different organs and summarizes freely his conclusions, only a few of which will be mentioned here. The most intense reflex influences upon the uterine movements were produced by irritation of the gastro-intestinal canal, especially of the colon. Stimulation of peristalsis by mechanical or chemical stimuli was accompanied by increased uterine contraction. Inhibition of peristalsis as produced by artificial distention of the stomach and segments of intestine produced likewise an inhibition of the uterine movements, a stage of rest with muscular relaxation. Division of the vagus or pelvic nerves produces a slight transitory inhibition of the uterine contractions; division of the sympathetics is accompanied by intense excitation. The afferent pathway for both inhibitory and stimulating impulses from the stomach and ileum is through the splanchnics; from the colon through the hypogastrics. The efferent section of the reflex arc is through both splanchnics and hypogastrics. The reflex centre for the entero-uterine connection lies in the prevertebral ganglia. Dilatation of the bladder and ureters leads to reflex inhibition, contraction of the bladder to reflex stimulation of the uterine musculature. Even after complete division of the hypogastric and pelvic nerves the vesico-uterine reflex persists, showing the presence of an independent reflex mechanism. Kehrer was unable to determine definitely the paths of the reflex between the breasts and uterus.

Genital Prolapse: Its Operative Correction Based on a New Study of Cleavage Lines and Sliding Segments.—R. L. DICKINSON (*Amer. Jour. Obst.*, 1910, lxii, 17) summarizes his article as follows: From the point of view either of pathological anatomy or surgery frozen sections show the importance of recognizing certain cleavage planes in the pelvic diaphragm, and intervening segments that slide. The cleavage runs (1) post pubic, close to the bones; (2) in the urethro-vaginal septum; (3) in the rectovaginal septum, and (4) along the anorectal canal.

If the urethral segment falls any considerable distance, only ventral fixation at the rear or top of the pubes will hold the upper urethra and anterior bladder wall. The second segment (vagina, bladder-base, cervix) is the common hernial mass. A convenient nomenclature would be intravaginal cystocele; extravaginal cystocele, protruding beyond the hymen on straining, and complete extrusion, bladder, cervix, uterus, one or all. For the worst cases Dickinson employs ventral fixation of the bladder, whether vaginal hysterectomy is done or not. The recto-anal segment, when very badly prolapsed, particularly in the presence of protrusion of the uterorectal pouch, may call for sigmoid fixation; and the wide-open long outlet of the pelvis may present no tissues out of which a diaphragm can be built, so that flaps from the buttocks may be required.

"Erosio" Portio Vaginalis Uteri.—ADAIR (*Surgery, Gynecology, and Obstetrics*, 1910, x, 337.) presents a brief resume of the various theories advanced to explain the origin of cervical erosions. In his opinion no individual theory embodies an explanation which can be applied to every erosion studied. From his studies of 57 cases he concludes that "erosions are chiefly inflammatory processes showing pictures which vary with the stimulus to their formation and the reaction of the tissue."

The Relation between Myoma of the Uterus and Conception.—OTTO GOETZE (*Ztschr. f. Geburtsh. u. Gynäk.*, 1910, lxvi, 340) includes the opinion of various writers concerning this subject and bases his conclusions upon the study of 105 cases. Small subserous myomas have no influence upon conception. With increasing size, the chances of conception are lessened, although not entirely excluded even by very large tumors. The prognosis varies with the site of the tumor, being most unfavorable in the submucous variety, especially when associated with extensive alterations in the endometrium and hemorrhages, as the result of increasing influence upon the endometrium, interstitial myomas lessen the likelihood of conception. Tumors of the cervix seem to offer greater obstacles than those of the corpus. If pregnancy does take place, myomas increase the danger of abortion. In the absence of other lesions which are conducive to sterility, myomectomy undoubtedly increases the chances of conception, the prognosis being more favorable the less radical the operation required, as in cervical and subserous myomas, the less the alterations in the endometrium, and the earlier the diagnosis is made. If myomectomy is not possible, an interstitial myoma of some size in a woman of thirty years practically excludes any hope of conception.

The Repair of Inaccessible Vesicovaginal Fistulæ following Hysterectomy.—G. G. WARD, JR. (*Surgery, Gynecology, and Obstetrics*, 1910, xi, 22) says that such a fistula is usually small, situated in the vault of an atrophic and contracted vagina, and embedded in the scar tissue which occupies the former position of the cervix, thus rendering operation for its cure extremely difficult, owing to its inaccessibility. The steps essential to cure by the method which he employs are: (1) The differentiation from a urethral fistula by distention of the bladder with an aniline solution. (2) Deep paravaginal incisions (Schuchhardt).

(3) A longitudinal incision of the anterior vaginal wall from the urethra to the vaginal vault through the fistula and extending into the posterior vaginal wall, and a lateral incision extending the full width of the vaginal vault. (4) Thorough separation of the base of the bladder from the entire vagina and vaginal vault. (5) Evisceration of the bladder into the vagina by means of a sound passed through the urethra. (6) Closure of the opening in the bladder separately from the vaginal incision. Suprapubic methods are to be employed only in the presence of failure by the vaginal route, since the latter is less dangerous and offers good chances for cure.

Gynecological Operations on Neurasthenics: Advantages, Disadvantages, Selection of Cases.—EDWARD REYNOLDS (*Boston Med. and Surg. Jour.*, 1910, clxiii, 113) defines neurasthenia as implying merely a state of low resistance to the depressing influences of environment, irrespective of the cause to which this low resistance is due. In attempting to ascertain the cause underlying these chronic conditions, with their manifold complaints, a careful history of the sequence of events, combined with an exact knowledge of the importance of symptoms, offers the best chances of success. In the presence of a demonstrable pelvic lesion, subsequent treatment demands a careful consideration of a possible chronological relation between this lesion and the symptomatology. In the absence of a reasonably close chronological sequence between general and local symptoms, local treatment should be withheld and general measures employed. When neurasthenia has developed secondary to a well-defined lesion, and if such a lesion is curable, operation is recommended. Such an operation should aim to repair rather than to remove the diseased organ, especially in women who have not reached the menopause.

Oliguria and Chronic Uremia in Carcinoma of the Uterus.—H. OFFEIGELD (*Archiv f. Gynäk.*, 1910, xci, 183,) states that chronic uremia is a frequent complication of inoperable carcinoma of the uterus, resulting from ureteral obstruction or from lesions of the kidneys produced by toxic products of the carcinoma. Should a palliative measure be deemed advisable to prolong life, nephrostomy, ureterostomy, or ureterocystoneostomosis will temporarily relieve the oliguria; the additional lesions of the kidneys, however, remain unchanged, thus exerting a deleterious influence upon the whole organism. Since the kidneys are supposed to possess an internal secretion, the author hoped to supply this substance and thus relieve the symptoms of chronic uremia by the administration of a renal extract obtained from the kidneys of beef. In four cases of carcinoma treated in this manner, the symptoms were exaggerated rather than relieved. He advances the idea that extracts of normal human kidneys may be useful, but so far this has not been determined. His conclusion is that animal renal extracts are valueless in alleviating the symptoms of chronic uremia associated with inoperable carcinoma of the uterus.

OTOLOGY.

UNDER THE CHARGE OF

CLARENCE J. BLAKE, M.D.,

PROFESSOR OF OTOLOGY IN THE HARVARD MEDICAL SCHOOL, BOSTON

The Prophylaxis of Deafness in School Children.—F. H. Quix (*Internationales Archiv f. Schulhygiene*, 1910, vi, No. 4) regards the examination of the ears of all school children, instead of its limitation to the selected cases in which an impairment has already manifested itself, as a matter of great importance. Hartmann's review of the proportion of cases of impaired hearing in school children, beginning with Bezold's classical communication on this subject in 1885, gives an average of 25 per cent. of pupils backward in their studies on account of this sense defect. Of this number very nearly one-half were capable of improvement under treatment (Bezold, 41.7 per cent.; Ostmann, 50 per cent.), but there are no statistics to show the proportion of cases in which the impairment of function could have been prevented by a rational prophylaxis, since, as yet, no provision has been made in the public schools for the more extended special examinations necessary to the institution of preventive measures. Regarding the study of causes as the proper basis for the institution of prophylactic measures, the author makes the following classification of etiological factors in the order of their causative importance: Adenoids, the exanthemas, typhus, parotitis, catarrhal and tuberculous processes in the nose and nasopharynx, hereditary syphilis, and traumas. The examination should include, in addition to the functional tests, an objective determination of existing abnormalities with reference to the past history, the present condition, and the prognosis as regards the resistance capacity; the detection of a disposition toward causative conditions being quite as important to the scholar as the determination of an actual impairment of hearing. The detection of even a moderate degree of tubal closure, for example, is of importance in the light of the changes which may take place in the sound transmitting apparatus, with frequent repetition or immediate continuance of this condition; while the detection of a perforation of the drumhead, consequent upon a past suppurative process, though the hearing may be so nearly normal as to suggest nothing through the subjective examination, is of importance in reference to the hygiene of the individual case; the residual evidences of middle-ear inflammation, cicatrices, atrophic areas, interstitial thickenings, and calcareous deposits in the drumhead are also evidences of a lessened capacity for resistance which should be taken into consideration. The demands of such a special examination are such as not only to take it out of the hands of the teachers, but to place it in those of a competent school physician whose professional acquaintance with the pupils should begin with their admission to the school, and be continued by repetition of the examination at stated intervals, or in individual cases, as occasion demands. The objective should precede the subjective examination, and for the

latter the main dependence is to be placed upon the hearing for the whispered voice, the words used being graded according to their inclusion of soft, medium, and loud consonant sounds. The advantage of this uniform hearing test for school purposes consists not only in its simplicity, in its accuracy of result, as compared with tests with tuning forks and acoumeters, but also in its international application, tests made by Delsaux, Reuter, Niedder, and Wojatschek, giving identical results in French, German, Italian, and Russian. The suggestions embodied in the author's paper are important for the consideration of all otologists in their bearing upon the furtherance of an adequate aural examination in public schools.

The Surgery of the Labyrinth.—GUSTAV ALEXANDER (*Archiv f. Ohrenheilk.*, 1910, lxxxi, Heft 3 and 4), in continuation of his communication at the International Otological Congress in Budapest, on the symptoms and treatment of inflammation of the labyrinth, reports a series of cases, and appends a classification defining the distinction between the complicated and simple cases of suppurative inflammation of the labyrinth. Endocranial implications, of otitic origin, are to be regarded as diseases for which surgery affords, in the great majority, the only assurance for recovery, since they present forms of suppurative inflammation of the meninges which, spreading rapidly, demand speedy operative interference; suppurative inflammations of the labyrinth, however, heal spontaneously in a not inappreciable percentage of cases. To this class belong: (1) The labyrinth inflammations secondary to suppurative cerebrospinal meningitis, in which the middle ear is unaffected and the labyrinth invasion has been effected through the medium of the internal auditory canal. (2) Otitic labyrinthitis, in which the infection is secondary to that of the middle ear, with suppurative implication of the membranous labyrinth, and formation of a labyrinth abscess, the bony capsule and surrounding petrous bone remaining intact. In these cases spontaneous healing may result as the sequence of a resorption of pus, the formation of connective tissue, and, later, osseous obliteration of the labyrinth, or evacuation of the labyrinth abscess into the middle ear through anatomical channels or by means of a fistulous opening. (3) Cases of suppurative labyrinthitis, secondary to middle-ear suppuration, through the medium of a fistulous opening through the tympanic portion of the osseous labyrinth capsule, the remaining portions of the bony labyrinth being intact, in which, unless there is cause for concern in the patient's general condition, spontaneous recovery from the labyrinth suppuration is to be expected, it being understood that recovery does not imply a restoration of function except in a very small percentage of cases, where circumscribed regions of the labyrinth have been spared from the suppurative invasion. A characteristic of all of these cases is the freedom from implication of all of the anatomical channels leading from the labyrinth toward, or into, the cranial cavity, the internal auditory canal, the aqueducts of the cochlea and of the vestibule, and, equally, the integrity of the surrounding barriers of the petrous portion of the temporal bone, including the tip, especially rich in diploetic cells, but having, between it and the anterior portion of the cochlea, an especially thick and compact capsular wall. These cases of otitic labyrinthitis, as contrasted with the cases of meningitic labyrinthitis, the

author classifies as uncomplicated labyrinth suppuration, cases in which, under conservative treatment, recovery is not infrequent. The complicated labyrinth suppuration, however, is one demanding operative interference, and that as speedily as possible upon the determination of symptoms of extension of inflammation to the cranial contents.

PATHOLOGY AND BACTERIOLOGY.

UNDER THE CHARGE OF

WARFIELD T. LONGCOPE, M.D.,

DIRECTOR OF THE AYER CLINICAL LABORATORY, PENNSYLVANIA HOSPITAL,

ASSISTED BY

G. CANBY ROBINSON, M.D.,

CLINICAL PATHOLOGIST TO THE PRESBYTERIAN HOSPITAL, PHILADELPHIA.

The Pathology of the Auriculoventricular Bundle of the Heart.—STERNBERG (*Central. f. allg. Path.*, 1910, xxi, 439) reports an examination in various parts of the bundle of His in 72 hearts of persons dying suddenly of cardiac failure. In about three-quarters of these cases various lesions were found in the conducting bundle, which consisted of hemorrhage, fatty infiltration or degeneration, diffuse inflammatory infiltration, small round-cell infiltration, with or without increase in connective tissue, and abscess formation. Usually the myocardium was affected in a manner similar to the conducting bundle, so that there was no ground for the assumption of an independent pathology for the His bundle. Sternberg thinks that the lesions of the bundle itself play a minor role in the production of sudden death, as disturbances of conduction are so often seen clinically without a fatal termination. The widespread disease of the muscle is, he considers, the causative factor in sudden cardiac death.

The Etiology of Hodgkin's Disease.—FRAENKEL and MUCH (*Münch. med. Woch.*, 1910, lvii, 685) have examined lymph nodes from cases of Hodgkin's disease by means of the antiformin method, with the result that they have found in 9 out of 10 cases a Gram-positive granular bacillus which resembles the tubercle bacillus in morphology. The material from the first two cases was obtained fresh. In the first instance the lymph nodes removed at operation were inoculated into guinea-pigs. At autopsy the picture of guinea-pig tuberculosis was found, with caseation of the mesenteric lymph nodes. In the organs the same Gram-positive granular bacillus was discovered, though there were no acid-fast bacteria. In the second guinea-pig, which was killed, the same condition was discovered. Material that had been recovered from eight other cases of Hodgkin's disease was studied by the antiformin method. In none of these cases had there been any tuberculosis at autopsy, but in seven of the eight cases the granular Gram-positive bacillus could

be demonstrated. These organisms were not acid fast. They were often present only in small numbers. Five cases of lymphatic leukemia were studied in the same manner, and in all of them Gram-positive granular bacilli with the morphology of the tubercle bacillus could be found in the lymph nodes and spleen. In the cases of lymphatic leukemia they were somewhat more numerous than in the cases of Hodgkin's disease. Fraenkel and Much conclude that the organism or group of organisms discovered by them bears a definite etiological relation to Hodgkin's disease and to lymphatic leukemia.

The Relation of Lymph Glands to Fat Metabolism.—STHEEMAN (*Ziegler's Beiträge*, 1910, xlviii, 170) has studied by various histochemical methods the distribution of fat in lymph nodes. He finds that in many animals as well as in man fat is constantly present in the lymph nodes under normal conditions. The fat is distributed in fine granules or is massed together in the protoplasm of the phagocytes. During digestion the mesenteric lymph nodes seem to play an important part in emulsifying and saponifying the fat on its transport from the intestinal wall to the thoracic duct. During periods of hunger or starvation the peripheral lymph nodes assume the same function toward the fat which is carried to the glands from the subcutaneous tissues. The phagocytes seem to exert an actual lipolytic action, for they not only emulsify the fat but probably exert some influence towards splitting it. In pathological conditions fat is also found in the phagocytes of the lymph nodes. During severe acute infections the lipolytic action seems to be decreased. In tuberculosis and even in lymphosarcoma of the lymph nodes the function of the glands toward fat metabolism is not destroyed. After subcutaneous injections of olive oil all the lymph nodes contain great quantities of partly unsaponified, partly saponified fats. Stheeman concludes that the lymph glands are organs one of whose functions it is to prepare fat for digestion. "Assimilation organs" he calls them, which are at the disposal of the nutritional fat as well as the tissue fat.

Compensatory Hypertrophy of the Small Intestine following Resection of Large Portions of the Jejunum and Ileum.—FLINT (*Trans. Conn. State Med. Soc.*, 1910, 283) has studied the effects of resection of large portions of the small intestine in 12 dogs, with special reference to a possible compensation on the part of the remainder of the intestine. He has found that as much as 50 per cent. or even 75 per cent. of the small intestine may be removed without causing death. At first there is severe diarrhœa, ravenous thirst and appetite, but recovery soon takes place, which may be only partial if three-quarters of the intestine is removed. Metabolic studies made upon three of these dogs by Erlanger and Hewlett showed that in these animals there was a marked increase in the excretion of the nitrogenous, fatty, and carbohydrate elements of the food. After compensation takes place digestion goes on as in a normal dog when a rich, easily assimilable diet is given, but should the fats of the food be greatly increased, the digestion is disturbed and the elimination of fats and nitrogen is increased to a point about 25 per cent. above normal. The compensatory mechanism consists in an hypertrophy of the villi of the remaining small intestines, which evidently represents an attempt on the part of the body to enlarge the absorbing

surface of the intestines. There is no actual increase in the number of villi, but such marked hypertrophy of the individual villi that in favorable cases the original epithelial surface of the intestine is restored by the compensatory hypertrophic process. In human beings the resection of large portions of the smal lintestine is a dangerous operation, for in 40 reported cases in which over 200 cm. of the small intestine has been resected, the mortality was 18 per cent. The human cases behave much like animals, though as yet no regeneration or hypertrophy has been reported. It is probable that patients who have had large portions of the small intestine removed would do best ultimately upon a rich and easily assimilable diet poor in fats and relatively rich in carbodydrates.

Experimental Typhus Fever.—NICOLLE, together with his assistants (*Ann. de l'Inst. Pasteur*, 1909, xxiv, 243), has investigated certain problems in regard to experimental typhus fever. After two unsuccessful attempts to transmit typhus fever to macaccus monkeys by inoculating these animals with blood from patients suffering with typhus, they succeeded in producing a typical attack of typhus of seven days, duration in a chimpanzee. The disease terminated in cachexia and death. On the day before the eruption appeared the blood of the chimpanzee was inoculated into a Chinese macacus and produced a typical attack of typhus. The blood of this monkey was found to be infective for the Chinese macacus, but not for other varieties of macacus tried. It was also non-infective for dogs and rats. Moreover, the preliminary inoculation of the Chinese macacus with blood from the human typhus seemed to confer upon this animal an immunity, for it was not suceptible to inoculation of blood of other macacus monkeys suffering from experimental typhus. The disease was transmitted to six macacus monkeys through the bite of fleas that had fed upon monkeys suffering from typhus. During the transmission of the disease from one generation of monkeys to another the virus appeared to decrease in virulence. The serum of convalescing monkeys was not harmless, but seemed to exert some definite toxic action when inoculated into healthy animals. The bacteriological examinations of the blood gave negative results. A study of the leukocytes, however, brought out several points of interest. During the early days of the attack, and beginning on about the fourth day, an extensive degeneration and often acutal necrosis of the polymorphonuclear leukocytes of the circulating blood was found, while later in the disease myelocytes and mononuclear cells appeared in considerable numbers.

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All communications should be addressed to—

DR. A. O. J. KELLY, 1911 Pine Street, Philadelphia, U. S. A.

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